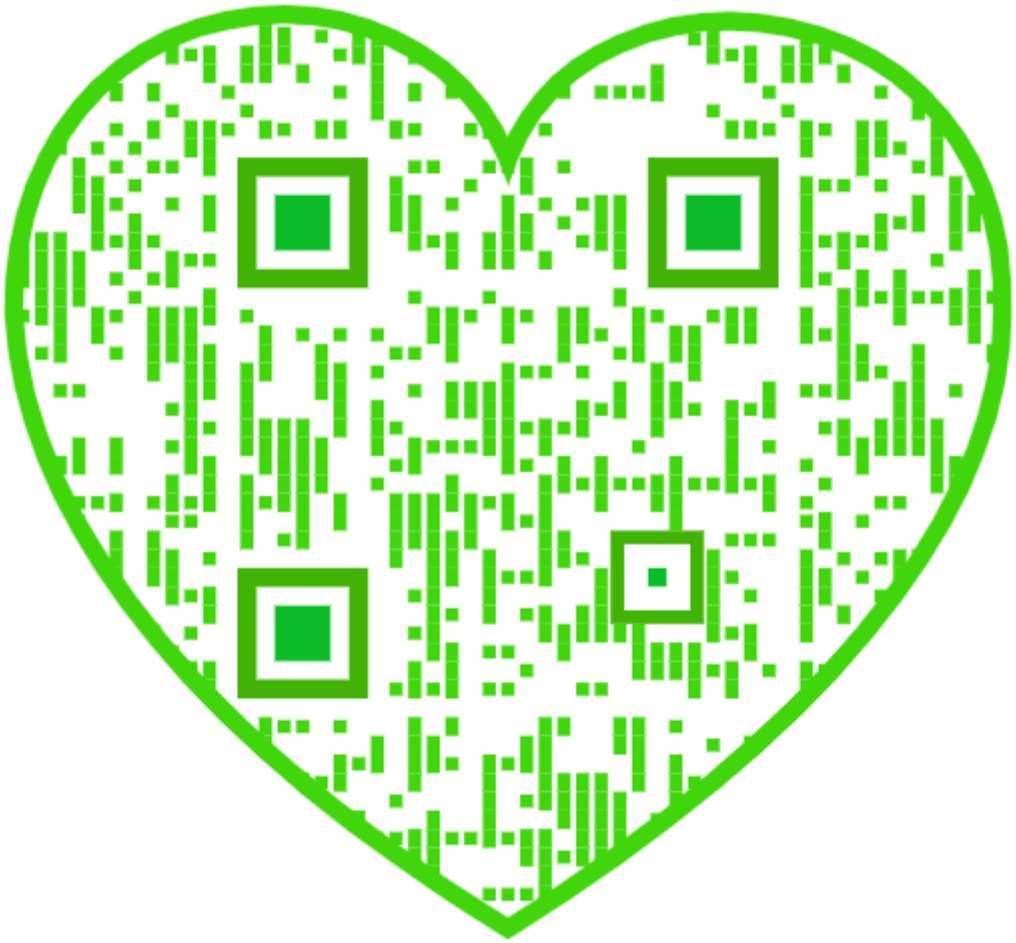

SURGERY OF THE UPPER ABDOMEN

DEAVER AND ASHHURST

SURGERY OF THE UPPER ABDOMEN

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TO THE
MEMORY OF

AGNEW
ASHHURST

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1921

PREFACE TO SECOND EDITION

The first edition of this work was published in two volumes: the first volume, comprising the surgery of the stomach and duodenum, appeared in 1909; and the second, which included the surgery of the gall-bladder, liver, pancreas and spleen, in 1913. So very favorable was its acceptance by the profession, that the publishers soon called for another edition; and work on the revision was well advanced when interrupted by the war. During the two years since the armistice the revision has been resumed.

While the general plan of the book remains unaltered, many sections have been entirely rewritten (*e.g.*, gastric ulcer, infantile stenosis of the pylorus, chronic dilatation of the duodenum, causes of death after operations on the stomach and duodenum, jejunal and gastro-jejunal ulcer, etc.), much new material added (*e.g.*, in the chapters dealing with operative technique, particularly the transgastric excision of ulcers, resection of the descending duodenum, cholecystectomy and operations on the bile-ducts, and the surgery of the spleen), and some that was obsolete omitted. The first edition contained extensive bibliographical references, most of which are of historic interest only, and therefore are not republished in this new edition. The dates of publication of papers of value, however, have been included in the text; thus the original references may readily be found, by those interested, in the Index Medicus or the Index Catalogue of the Library of the Surgeon General's Office, United States Army. Statistical tables published at length in the first edition have been condensed or summarized, and whenever possible have been superseded from the growing statistics available especially from the senior author's service at the Lankenau Hospital. Moreover, by the use of a slightly larger page, considerable space has been gained, so that it is now possible to present in a single volume of slightly over 800 pages more material than was formerly included in two volumes running almost to a thousand pages.

Much consideration has been given to the matter of illustrations; nearly one hundred new drawings have been introduced, most of them from the skilful pen of Mr. E. F. Faber, and many of them from preparations by the junior author.

Analysis of the literature which has appeared during the last ten years has been no light task; and without the intelligent and capable assistance of Miss A. M. Jastrow it could not have been accomplished. All the references have been verified by the junior author. Thanks are also due to Miss Edna Patterson for her continued assistance in preparing the manuscript for the press. Dr. A. D. Whiting has again consented to prepare the Indices, and has fulfilled his undertaking in a manner that adds inestimably to the value of the volume.

J. B. D.
A. P. C. A.

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SURGERY OF THE UPPER ABDOMEN

CHAPTER I

ANATOMY

Systematic descriptions of anatomy are sought in treatises specially devoted to the subject; and what a reader desires to find in a work like the present is the surgical or applied anatomy of the parts under discussion. The histological structure of the various organs is therefore omitted, together with a detailed statement of their size, shape, and minute relations to other structures. It has seemed wise, moreover, to consider the subject of the anatomy of the upper abdomen as a whole, and not to preface the surgery of each organ with a brief anatomical outline in which there would be many repetitions required.

The Abdominal Wall.—To the operating surgeon the abdominal wall should present itself as a muscular and aponeurotic structure whose subsequent strength depends upon the skill and judgment with which it is divided and sutured during an operation. The abdominal muscles, like muscles elsewhere in the body, are supplied with arteries, veins and nerves; and the incision through these muscles should be made with due regard for their preservation, especially for preservation of the nerves, since injury to the latter is permanent and irreparable.

The rectus muscle is attached at the costal margin as far outward as the ninth costal cartilage. In its upper-two-thirds the rectus muscle is about 8 cm. broad, being somewhat narrower as it approaches the pubic spine. In the cadaver the muscle is usually less broad than during life, often measuring less than 5 cm. in width. Outside of the semilunar line the incisian should be transverse, and more or less oblique; the fibres of the lateral muscles of the abdomen do not all run in the same direction, and any simple incision must divide one of the muscular planes obliquely. Yet after transverse division of any muscle, accurate suture will restore almost its pristine integrity, so long as its nerve supply has been preserved. Transverse division even of the rectus muscle, though undesirable, results, after careful suture, merely in adding one more *linea transversa* to its structure.

The chief artery met with in the upper abdominal wall is the in-

ternal mammary or some of its terminal branches. It runs between the rectus muscle and its posterior sheath, and the larger branches are toward the middle line of the body. The lower intercostal nerves run forward transversely between the internal oblique and transversalis muscles (Fig. 1), pierce the posterior sheath of the rectus muscle

FIG. 1.—Dissection of the Right Half of an Abdominal Wall. The External and Internal Oblique Muscles Have Been Turned to the Left, Exposing the Lower Intercostal Nerves and the Deep Circumflex Iliac Artery, as They Lie upon the Transversalis. From a Dissection by the Junior Author in the Laboratory of Operative Surgery in the University of Pennsylvania.

(the deep lamella of the aponeurosis of the internal oblique), and enter the rectus muscle from its deep surface (Fig. 2). They will be cut by an incision parallel to the fibres of the rectus, unless this

incision is made close to the linea alba; and loss of contractility and atrophy of the part of the muscle so affected follows such injury; but as a rule the impairment of function in the epigastric region is less serious than in the hypogastric region.

The distribution of the lower intercostal nerves is important in connection with cutaneous hyperesthesia, muscular rigidity, and referred pain—all conditions frequently encountered in abdominal affections. The sixth and seventh nerves supply the skin in the epigastric region (the "pit of the stomach"); the eighth and ninth, that region between the epigastrium and the umbilicus; and the tenth, the umbilical area. The cutaneous hyperesthesia, referred pain, and muscular rigidity of the abdominal wall are due, as is well known, to the overflow of the stimulation received by the cells in the spinal cord from the diseased area within the abdomen. When the stimulation overflows into sensory nerve filaments, cutaneous hyperesthesia and pain are produced; but the motor nerves are usually affected also, and hence muscular rigidity of the overlying abdominal wall is produced (viscero-muscular reflex of Mackenzie), by the same mechanism as that by which, as was long ago pointed out by Hilton, an inflamed joint is held rigid by its enveloping muscles. As the flat muscles of the abdominal

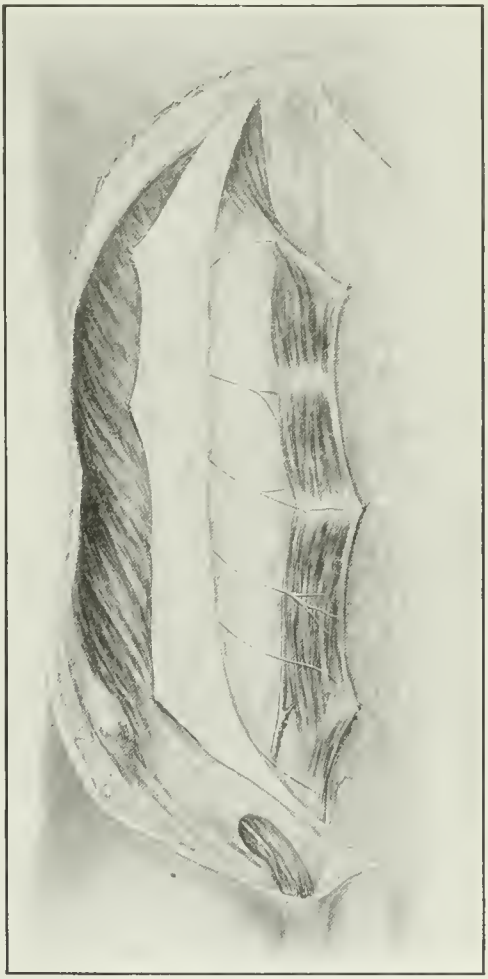

FIG. 3.—The Six Lower Intercostal Nerves and the First Lumbar Nerve Entering the Rectus Muscle from its Deep Surface. The Rectus Muscle Has Been Drawn from Its Sheath and Turned toward the Left. From a Dissection by the Junior Author in the Laboratory of Operative Surgery in the University of Pennsylvania.

wall are not innervated by a single nerve trunk, but by numerous twigs from different nerve trunks, where the viscero-muscular reflex is referred along one trunk only, merely a portion of the muscle will contract. This is not so important in the upper abdomen as in the iliac region, where such band like rigidity has been mistaken for a palpably enlarged appendix.

The pain referred to the left shoulder blade in disease of the duodenum and stomach is to be explained by the connection between the pneumagastric nerves and the sympathetic ganglia on the left side. Mayo Robson and Moynihan have pointed out that as long as the gall-bladder only is involved, the referred pain is felt in the right infrascapular region, but as soon as the inflammation or adhesions involve the pylorus, the pain is felt also in the left infrascapular region. Pain may be referred to the neck from involvement of the phrenic nerve, and to the shoulder tip through its connection with the supra-acromial nerve (p. 26).

Running from the umbilicus upward, along the deep surface of the right rectus muscle, to the longitudinal fissure of the liver, is the falciform or suspensory ligament of this organ, containing between its layers the round ligament or obliterated umbilical vein of the fetus; as well as some small veins from the epigastric vein, anastomosing with the portal system; some arterial twigs from the phrenic arteries; besides lymphatics and nerves. It is advisable, therefore, if this fold of peritoneum must be divided when making incisions through the abdominal wall to be careful that no oozing of blood goes undetected. The suspensory ligament lies rather close to the linea alba, and hence an incision through the outer half of the right rectus muscle will not injure it; if an incision just to the right of the linea alba is to be extended past the umbilicus, care should always be taken to check any bleeding from the structures in the round ligament of the liver.

Surface Anatomy.—The *umbilicus* is at the level of the third lumbar vertebra. Approximately between it and the spinal column lies the third or transverse portion of the *duodenum*. Cephalad to the umbilicus in the recumbent patient lies the *transverse colon*, about three fingerbreadths wide, and between this and the ensiform cartilage are found the pyloric portion of the stomach, and, overlapping this, the left lobe of the liver. The longitudinal fissure of the liver, separating the left from the right lobe, is less than 2.5 cm. to the right of the median line of the body. If the transverse colon sag, and hang below the umbilicus, some coils of small intestine may present themselves between the colon and the stomach, displacing the transverse mesocolon, which,

as well as the gastro-colic omentum, will be found interposed between these displaced intestines and the anterior abdominal wall.

The central tendon of the *diaphragm* is found at the base of the ensiform process of the sternum, at the level of the cartilage of the sixth or seventh rib, and opposite the eighth dorsal vertebra. The lateral arches of the diaphragm rise and fall slightly during respiration, the right being slightly higher than the central tendon, and about 2 cm. higher than the left arch.

The *liver* fills the right hypochondriac region, and extends through the epigastrium to the left hypochondriac region to a distance of from 2 to 4 cm. beyond the left border of the sternum. It may reach the left mammary line. The liver extends as high as a transverse line drawn through the lower end of the gladiolus (the mesosternum), or the base of the ensiform cartilage. The upper surface of the left lobe is on this same level (the fifth intercostal space); but the right lobe is a trifle higher and is said to reach the lower border of the fifth rib. Since the position of the liver varies slightly with that of the body, and with the movements of the diaphragm, these outlines are only approximately correct. The lower surface of the right lobe of the liver posteriorly is opposite the spine of the eleventh dorsal vertebra, and in the midaxillary line is at the costal margin; between the mid-axillary line and the right semilunar line the thin anterior margin of the liver projects about 2 cm. below the costal margin, and crosses the median line of the body in a line drawn from the ninth right, to the eighth left, costal cartilage. The gall-bladder lies beneath the ninth right costal cartilage in the semilunar line, at the outer border of the right rectus muscle.

The relations of the *stomach* vary considerably. When distended, it is in contact with the anterior abdominal wall in a triangle bounded by the anterior margin of the liver, the left ninth and tenth costal cartilages, and a line drawn between the tenth costal cartilages. The cardiac orifice of the stomach is opposite a point 2.5 cm. to the left of the seventh left chondro-sternal junction, at the level of the eleventh thoracic vertebra; the pyloric orifice lies beneath the liver, about 3 cm. below the base of the ensiform cartilage, at the level of the upper edge of the first lumbar vertebra; but as the stomach becomes distended the pylorus approaches the right linea semilunaris. The line for the lesser curvature of the stomach is drawn from the position of the cardiac orifice to that of the pylorus. The line for the greater curvature extends upward and to the left from the position of the cardiac orifice to the fifth rib, slightly external to the left mammary line, and thence

to the position of the pyloric orifice. The line of the greater curvature is convex downward and to the left; that of the lesser curvature is nearly vertical when the stomach is empty.

The *duodenum* is from 25 to 30 cm. in length, commencing at the pyloric orifice of the stomach, and ending in the jejunum at the left side of the body of the second lumbar vertebra, after having described a half circle with its convexity downward. The first portion of the duodenum, about 5 cm. long, passes from the pyloric end of the stomach to the right, upward and backward, to the neck of the gall-bladder; the second portion is about 8 cm. in length, and extends from the neck of the gall-bladder, downward along the right of the spinal column to the level of the third lumbar vertebra. Here the third portion of the duodenum commences, passing obliquely upward to the left, across the body of the second lumbar vertebra. A line drawn from a point 8 cm. to the right of the umbilicus, to a point 5 cm. to the left and above it, will nearly indicate the position of the third portion. The fourth portion is only about 2 cm. in length; it passes upward from the termination of the third portion, and becomes continuous with the jejunum at the origin of the mesentery.

The *pancreas* extends across the bodies of the first and second lumbar vertebræ from the hilum of the spleen in the left hypochondriac region, to the second portion of the duodenum in the epigastric region. Its length is from 15 to 20 cm., and its largest portion, called the head, is surrounded by the semicircle of the duodenum as a picture is surrounded by its frame; while its body crosses the spinal column, and its tail is in contact with the spleen. It lies between the celiac axis, above, and the superior mesenteric vessels, below, these latter separating it from the transverse (third) portion of the duodenum.

The *spleen* extends in the mid-axillary line longitudinally from the ninth to the eleventh rib, and transversely from the midaxillary to the posterior axillary line. Its long axis is nearly parallel to the course of the ribs.

Embryology.—To understand the various folds and recesses of the peritoneum in the upper abdomen it is essential to revert to the embryonal stage before this membrane has developed the perplexing conditions found in adult life. For practical purposes it is sufficient to describe the fetal state as follows: The peritoneum is to be regarded as a closed sac filling the abdominal cavity; along the posterior part of the abdominal cavity, back of this closed sac and parallel with the spinal column, runs the digestive tract in the form of a long straight tube. At first this tube is in connection with the region outside of the

abdominal cavity by means of a prolongation through the navel, known as the vitelline duct. This duct later becomes detached from the navel, but is sometimes still evident in adult life as Meckel's diverticulum. While still attached to the umbilicus it acts as a guy rope, and pulls the formerly straight intestinal canal forward in a **U**-shaped projection, the arms of the **U** being known as the upper and lower (Fig. 3). When the intestinal tube is thus pulled forward, the

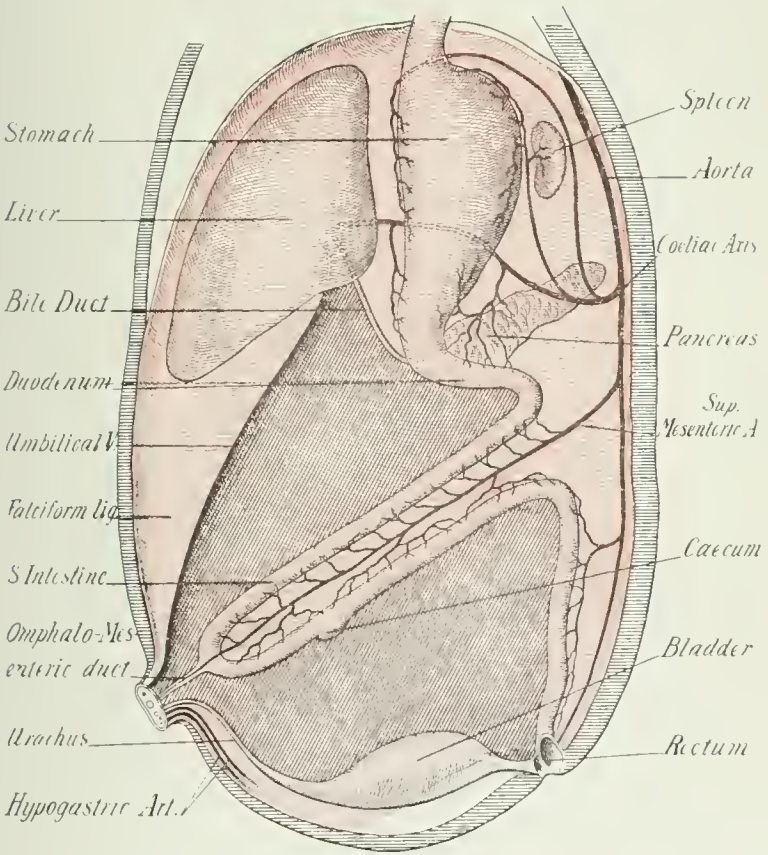

FIG. 3.—Diagram of Early Stage of Development. The Portions of the Parietal Peritoneum not Drawn Away from the Body Walls to Form Mesenteries, Ligaments, etc., are Indicated by Deeper Shading.

closed peritoneal sac is pushed in front of it by the intestine, which becomes more or less completely covered by the peritoneum, still retaining, however, an extraperitoneal surface through which it receives its blood vessels, nerves and lymphatics. The two folds of peritoneum covering these structures, as they pass to the intestinal tube, are known as the mesentery. The upper part of the primitive intestinal tube,

close beneath the diaphragm, becomes dilated, and forms the stomach at first it lies longitudinally in the abdominal cavity, and somewhat resembles the bulb of a hand syringe in its relation to the rest of the tube. Its greater curvature lies posterior, and the pylorus is its lowest part. That portion of the primitive intestinal tube just below the stomach forms the duodenum, and from its anterior wall the liver grows out, as a compound tubular gland. The rapid growth of the liver, and its position close beneath the diaphragm, account in large measure for the peculiar distributions of the peritoneum around it. It develops from the anterior surface of the duodenum, and grows forward, pushing the peritoneum in front of it and downward, leaving a pedicle of peritoneum only at its origin from the duodenum and along its inferior surface, lying, so to speak, above the closed peritoneal sac, between this membrane and the diaphragm. From the umbilicus there passes upward outside the anterior wall of the closed peritoneal sac, a vein, known as the umbilical vein, which persists in the adult as the round ligament of the liver, and which is enveloped by two folds of peritoneum known as the falciform or suspensory ligament of the liver. The urachus and the hypogastric arteries raise similar folds of peritoneum below the umbilicus. The round ligament passes to the longitudinal fissure of the liver, and when it reaches the transverse fissure blends with the portal vein, into which vessel it emptied its blood during intrauterine life.

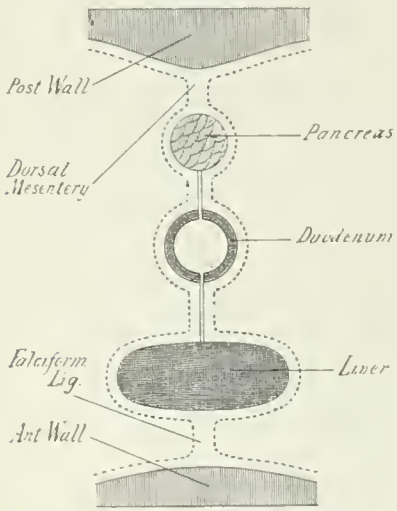

FIG. 4.—Diagram Showing Relation of Duodenum to Liver and Pancreas.

The duodenum at this period of development has already a mesentery of its own, slight in extent, lying between it and the posterior abdominal wall, and of course continuous above with the gastric mesentery and below with that of the jejunum. Into the layers of this duodenal mesentery the pancreas grows, extending backward from the duodenum, just as the liver grows forward (Fig. 4).

Now commences a complex process of rotation of all the abdominal viscera. The lower limb of the U-shaped intestinal tube, in which the cecum begins to bud, rotates upward in front of and above the upper

limb, and the cecal portion passes first through the umbilical region to the left hypochondriac region, thence to the right hypochondrium, and finally at birth settles down toward the right iliac region of the abdomen. This rotation of the intestine takes place from left to right around the superior mesenteric artery as an axis in such a manner that the colon crosses the commencement of the small intestine transversely. While in this way the commencement of the large intestine

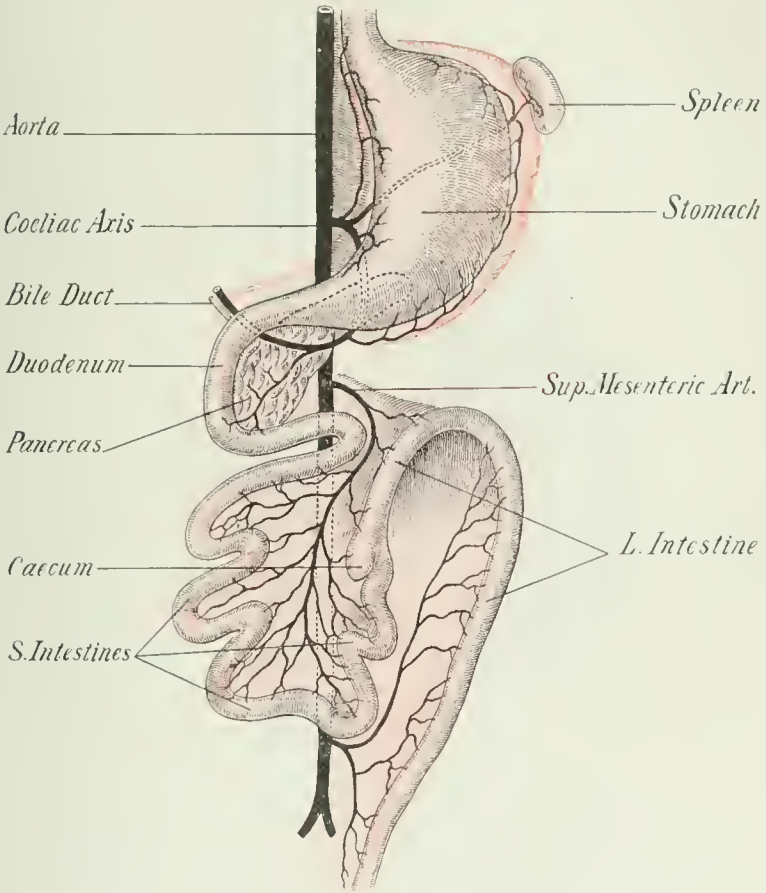

FIG. 5.—Diagram Representing Early Stage of Rotation of Abdominal Viscera.

is thrown over to the right side, the small intestine for the greater part assumes a position on the left, and the former right side of the mesentery becomes the left and *vice versa* (Fig. 5). Thus, the lower part of the duodenum is carried to the left and the commencement of the large intestine is carried across it—an explanation of the position of the duodenum behind the transverse colon in the adult, and of the

passage of the superior mesenteric artery over the front of the duodenum. The influence that the rotation of the intestinal loop has upon the mesentery may be readily appreciated: the attachment of the mesentery of the small intestine (the upper limb of the U-shaped digestive tube) remains practically unchanged, while that of the large intestine assumes attachments corresponding to the ascending, the transverse and the descending mesocolon. At the same time that this intestinal rotation is taking place from left to right, the

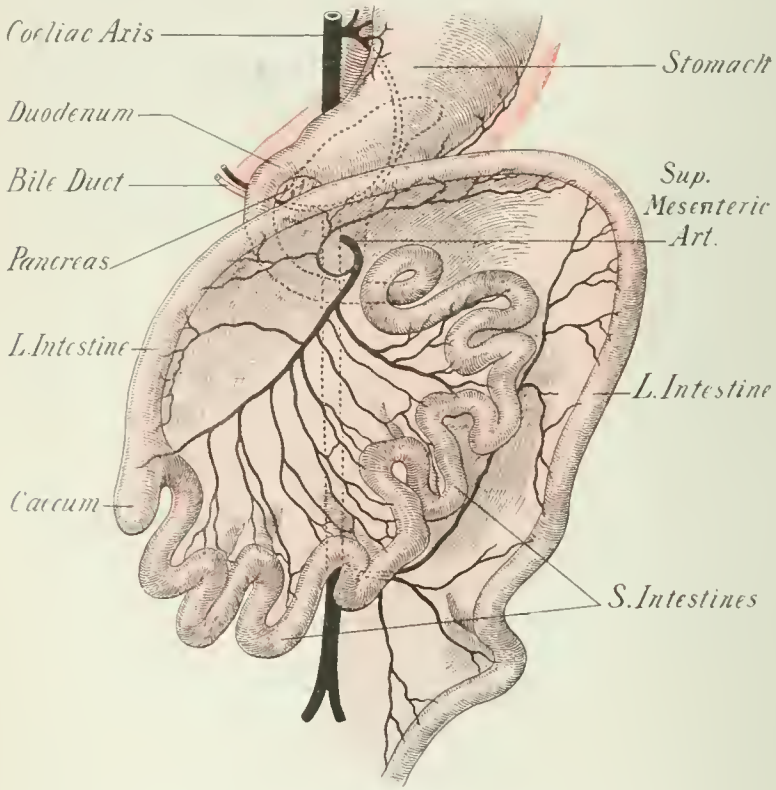

FIG. 6.—Diagram Representing Later Stage of Rotation of Abdominal Viscera.

stomach likewise undergoes rotation in the same direction, so that its left side becomes anterior, and its right side posterior in position. The spleen, which from about the fifth week of intrauterine life may be distinguished in the dorsal mesogastrium, is carried to the left by the rotation of the stomach; and the gastro-splenic ligament corresponds to that portion of the dorsal mesogastrium in which it originally developed. The liver passes to the right hypochondriac region, and the pancreas is shifted slightly to the left. The pyloric end of the

stomach ascends, and the greater curvature becomes the inferior border, while the lesser curvature becomes the superior border of the stomach. The stomach has now therefore an anterior and a posterior wall, both covered with peritoneum; but whereas the anterior is in free communication with the general cavity of the peritoneum, the posterior wall has become more or less isolated, and is in relation with the pancreas, the lesser peritoneal cavity separating them (Fig. 6). This lesser peritoneal cavity retains its only connection with the general peritoneal cavity at its right extremity through the foramen of Winslow. Above the stomach the gastro-hepatic omentum stretches from its lesser curvature to the liver, while from its greater curvature the great omentum passes downward between the stomach and the transverse colon, at first consisting of two double folds of peritoneum. Later these double folds fuse and become adherent to the transverse colon, so that the adult type is found shortly after birth. The duodenum and the pancreas are by this process of rotation sequestered behind the stomach and transverse colon, and being subject to no movement of any consequence lose their posterior mesenteries by absorption, and become in extrauterine life retroperitoneal organs. It is a law that when two serous surfaces are approximated, and little or no motion exists between them, they fuse. Thus, the duodenum and its mesentery, in which the outgrowth of the pancreas develops, are pressed by the transverse colon against the posterior abdominal wall and unite extensively with the peritoneum covering the latter. The mesentery of the small intestine, which grows apace with the gut at its intestinal attachment, is thrown into fan-shaped folds, since at its vertebral attachment it remains short. The transverse mesocolon, carried by the colon transversely across the end of the duodenum, obtains secondary attachment to the latter and to the posterior abdominal wall, in a line from left to right, and remains permanently as a well-marked mesentery. Thus, the transverse colon with its mesocolon divides the abdominal cavity into an upper part that includes stomach, liver, duodenum and pancreas, and a lower which contains the small intestine. The mesenteries of the ascending and descending colon become obliterated by fusing with the parietal peritoneum of the posterior abdominal wall, so that in the mature condition these parts of the gut are, as a rule, covered by peritoneum only in front and at the sides.

The development of the great omentum begins in the third month of fetal life. Being originally the posterior mesogastrium, attached at the greater curvature of the stomach, it extends gradually downward

above the transverse colon. Coming in contact in the first part of its course with the transverse mesocolon, the great omentum soon fuses with this, and subsequently with the transverse colon, and this relation becomes permanent; their bloodvessels, however, remain distinct: the gastro-epiploic arteries supply branches to the stomach and omentum, while the transverse colon is supplied by the middle colic artery. This provides relatively avascular areas through which the posterior wall of the stomach may be exposed, by detaching the great omentum from the transverse colon—the so-called intercolo-epiploic route (Fig. 11). The pancreas, at first situated between the two layers of the mesogastrium, acquires its retroperitoneal position also during the third month.

Topographical Anatomy. Stomach.—We may recognize two main divisions of the stomach, the *cardiac* and the *pyloric portions* (Fig. 7),

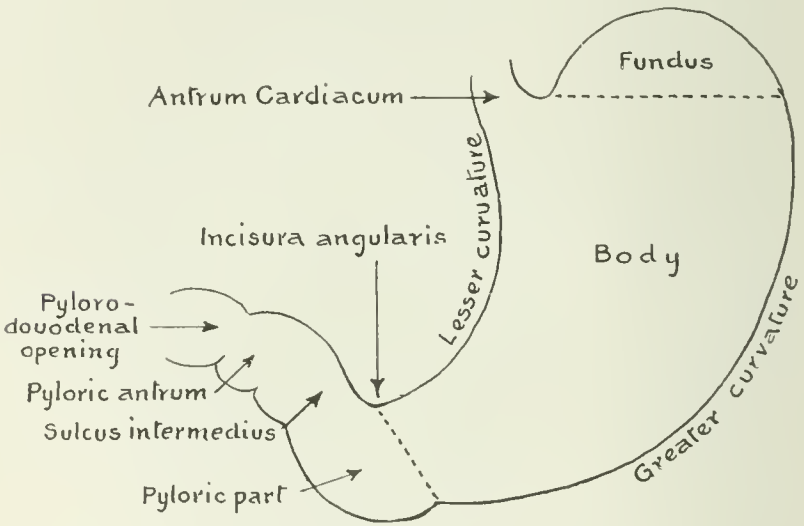

FIG. 7.—Diagrammatic Outline of the Stomach.

which are separated by the *incisura angularis*, at the point where the lesser curvature at first nearly vertical changes its direction to run upward and backward. The uppermost part of the cardiac portion is called the *fundus;* this lies wholly above the level of the cardiac orifice. The *cardiac antrum* is that part of the gastric lumen which corresponds to the abdominal part of the esophagus. The *pyloric antrum* is the terminal 2.5 cm. of the gastric canal, immediately adjacent to the pylorus; it is limited toward the patient's left by the *sulcus intermedius*.

The musculature of the pyloric portion of the stomach is much

more developed than is that of the body of the stomach, a fact which is explained by the motor functions of the stomach during digestion (p. 46). When tonic contraction of the pyloric portion exists, it may bear considerable resemblance to the outline of an hourglass stomach (Fig. 23). The cardiac orifice of the stomach is directed almost horizontally toward the left, so that the surgeon's finger, seeking entrance to the esophagus from within the stomach, must be passed toward the patient's right.

In adult life the stomach is almost entirely intraperitoneal. It retains its primitive mesentery from its greater curvature in the form of the gastro-colic omentum; while the mesentery acquired for it by the growth of the liver, attached to the lesser curvature, is known as the gastro-hepatic omentum. The greater and lesser curvatures of the stomach are thus extraperitoneal, and contain the main *blood vessels*. Along the lesser curvature run from left to right the gastric or coronary artery, from the celiac axis, and from right to left the pyloric artery, from the hepatic artery, itself a branch of the celiac axis. Along the greater curvature of the stomach runs from left to right the gastro-epiploica sinistra, from the splenic, and from right to left the gastro-epiploica dextra, from the hepatic through the gastro-duodenal. The anastomosis of both pairs of arteries is very free, and when divided at any part of their course severe hemorrhage from both ends is to be anticipated. Smaller branches are given off at right angles, which run transversely across the walls of the stomach. The branches from the lesser curvature supply about two-thirds of the areas on the anterior and posterior gastric walls. The veins correspond to the arteries, and ultimately empty into the portal vein. The left gastro-epiploic usually is the main source of the blood supply of the omentum, ramifying on its posterior surface; while the right gastro-epiploic is distributed more to the stomach.

Except for these omental regions the only *extraperitoneal* portion of the stomach is a small and irregular triangular area on its posterior surface near the cardiac opening. One angle of this triangle is at the point where the coronary artery reaches the stomach (the gastro-phrenic ligament), a second is at the commencement of the gastro-splenic portion of the great omentum, while the third is to the left of and below the cardiac opening of the stomach.

The *lymphatics* of the stomach are of considerable importance in connection with the metastasis of malignant growths, and have only within recent years received adequate attention. The stomach may be divided roughly into three lymphatic areas (Fig. 8): one, in the region

of the fundus of the stomach, where the nodes are few, and two others, along the greater and lesser curvatures respectively (Cunéo, 1900). Of these latter two areas, the nodes along the lesser curvature are much more apt to be involved in malignant growths, the area affected extending as far toward the esophageal end of the stomach as the position of the coronary artery; while the duodenum is rarely involved for a distance of more than two or three centimetres. The practical deductions from these facts will be considered in greater detail when discussing malignant growths of the stomach. From these various lymphatic nodes the lymph vessels pass to the celiac nodes; the vessels from the lesser curvature following the course of the coronary artery, and those from the greater curvature running with the right gastro-epiploic vessels, both sets eventually meeting in the same nodes (celiac) around the aorta, above the origin of the superior mesenteric artery. Jamieson and Dobson have made a more recent study of the lymphatics of the stomach (1907). They found nodes beneath the pylorus quite frequently present, draining the neighboring portion of the greater curvature. In not a few instances they were able to trace lymph channels from the pylorus directly past the lower coronary group of glands into the right suprapancreatic glands lying along the trunk of the hepatic artery.

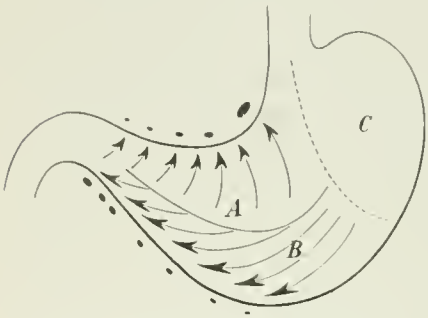

FIG. 8.—Lymphatic Areas of the Stomach.

The stomach is supplied liberally by sympathetic *nerves*, as well as by the terminal filaments of the pneumogastric. The left pneumogastric curves around to the anterior border of the esophagus, just above the cardiac orifice of the stomach, and distributes branches to the lesser curvature and anterior wall of the stomach; while the right pneumogastric is similarly distributed over the posterior wall. Filaments from both nerves inosculate along the greater curvature. Some filaments from the right nerve pass to the left side of the celiac and splenic plexuses of the sympathetic system, while some of the filaments from the left nerve pass from the lesser curvature of the stomach through the gastro-hepatic omentum to the hepatic plexus.

The posterior wall of the stomach cannot be satisfactorily palpated through the foramen of Winslow; as a rule only the posterior surface of the pylorus is thus reached (Fig. 9). In the free fold of the gastro-hepatic omentum may be felt the common bile duct, furthest forward,

FIG. 9.—Probe in Foramen of Winslow. The Liver Has Been Drawn Upward to Expose the Gastro-hepatic Omentum.

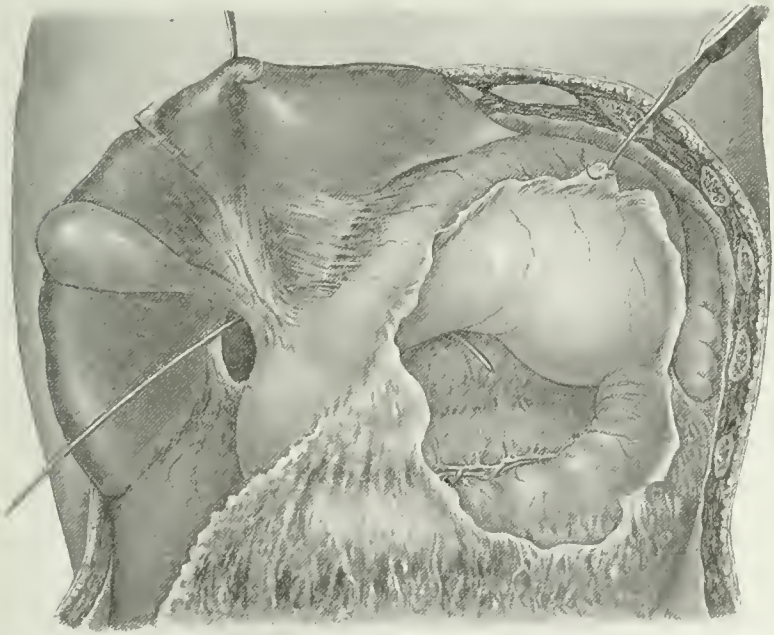

FIG 10.—Structures in the Lesser Peritoneal Cavity Exposed by Dividing the Gastro-colic Omentum.

and further in and to the patient's left the hepatic artery, with the portal vein behind and between. The duct of Wirsung (pancreatic) is too short and too low down to be palpated without loosening the layer of peritoneum covering the right side of the descending duodendm. To

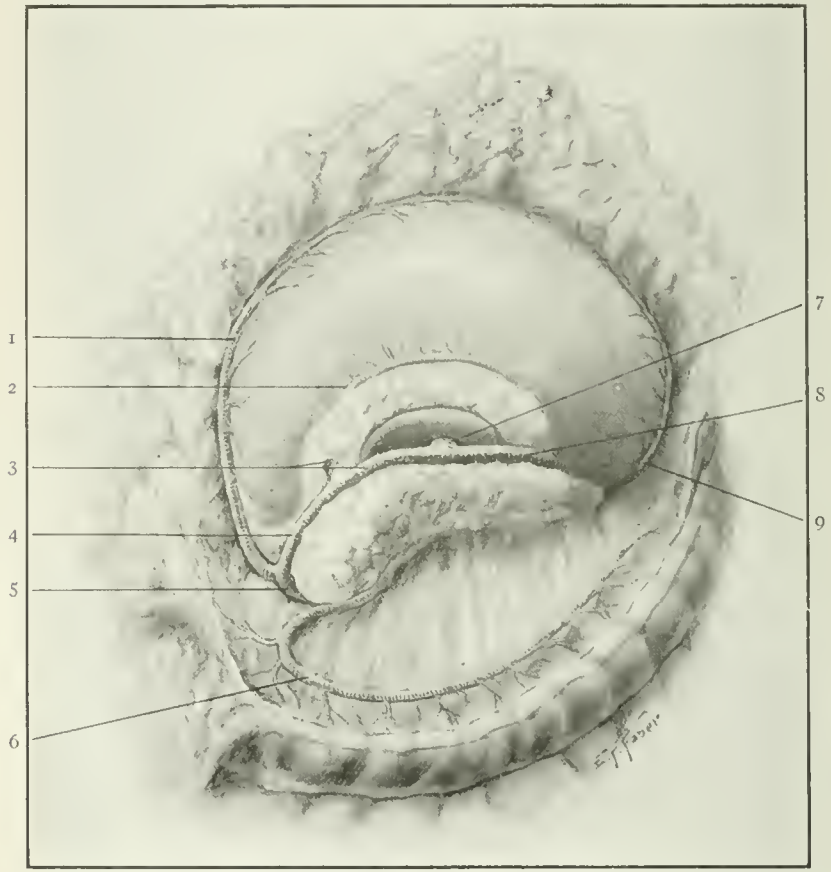

FIG. 11.—Posterior Gastric Wall Exposed by Detaching Great Omentum from Transverse Colon—the "Intercolo-epiploic Route." The Stomach Has Been Turned Upside Down Around its Lesser Curvature as a Hinge, Freely Exposing the Lesser Peritoneal Cavity.

1. Right gastro-epiploic artery.
2. Gastric or coronary artery.
3. Hepatic artery.
4. Gastro-duodenal artery.
5. Superior pancreatico-duodenal artery.
6. Middle colic artery.
7. Celiac axis; above it the Spigelian lobe of the liver.
8. Splenic artery; below it, the pancreas.
9. Left gastro-epiploic artery.

From a Dissection by the Junior Author in the Laboratory of Operative Surgery in the University of Pennslyvania.

expose thoroughly the posterior wall of the stomach we have a choice of three routes—through the gastro-hepatic omentum, through the gastro-colic omentum, or by detaching the great omentum from the

transverse colon, thus passing below the great omentum. Only in cases with marked ptosis of the stomach is adequate exposure obtained through the gastro-hepatic omentum. Where, however, the gastro-colic omentum is sufficiently wide to permit, this may be divided, close to the colon, so as to avoid the gastro-epiploic arteries; the stomach is then partially inverted through the opening (Fig. 10). But a still wider exposure, with no damage to any bloodvessels of consequence, is obtainable through the third route, namely by passing beneath the great omentum and detaching it from the transverse colon (Fig. 11). Access across the transverse mesocolon, sufficient for the performance of posterior gastro-jejunostomy, is not sufficient for adequate exploration.

Liver.—The liver presents several *extraperitoneal areas*. The largest is on the postero-superior surface of the right lobe, between the layers of the right portion of the coronary ligament (Figs. 12 and 15). Here, about the middle of the posterior surface of the liver, the inferior vena cava is found. The extra-peritoneal area between the layers of the median and left portions of the coronary ligament is insignificant in size, as is also that region about the transverse fissure where the bile duct, the portal vein and the hepatic artery are found. For practical purposes, therefore, the liver is wholly an intraperitoneal organ; although abscesses pointing through its superior surface are usually excluded from the general peritoneal cavity by adhesions.

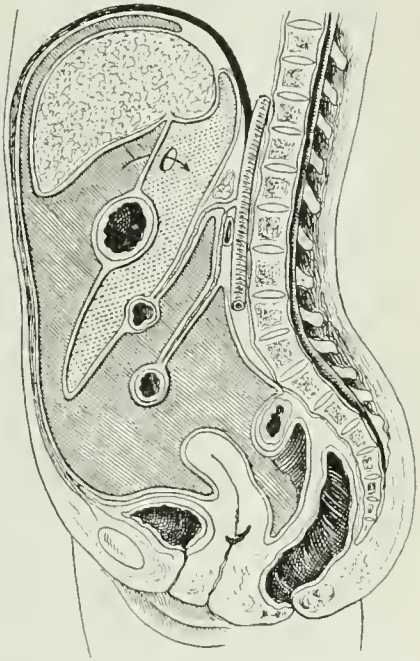

FIG. 12.—Diagram of Peritoneal Reflections in Median Sagittal Section. Note the Arrow in the Foramen of Winslow; the Stomach Suspended from the Liver by the Gastro-hepatic Omentum; and the Superior Mesenteric Artery Emerging from the Aorta Between the Pancreas and Duodenum, and Giving off its First Large Branch, the Middle Colic Artery.

When the hand is introduced between the right lobe of the liver and the diaphragm through an abdominal incision, it passes backward over the upper convex surface of the liver for about 15 cm., when the finger tips are arrested by the coronary ligament, running transversely across the surface of the liver. The falciform ligament

will be felt running forward from the coronary ligament, close to the median line of the body, and will prevent the fingers from passing from the surface of the right lobe across to that of the left. By carrying the hand well along to the right edge of the liver, the right extremity of the coronary ligament, known as the right lateral ligament, will be felt, and in some cases the fingers can be passed around the free margin of this ligament on to the posterior surface of the liver, back of the posterior layer of the coronary ligament. The hand is here arrested by the reflection of the visceral peritoneum on to the posterior abdominal parietes. On the left side of the falciform ligament, above the left lobe of the liver, the left coronary ligament, and its extreme portion, the left lateral ligament, may likewise be palpated by the examining hand.

The under surface of the liver is also quite easily examined by the sense of touch. At the cartilage of the right ninth rib, about 5 cm. to the right of the falciform ligament, held close against the under surface of the liver by a fold of peritoneum, is the gall-bladder, and by following this landmark backward with the fingers, we are led first to the cystic duct, then across the anterior margin of the foramen of Winslow along the common bile duct in the free margin of the gastro-hepatic omentum, to the posterior surface of the duodenum. Beyond this point the bile duct usually cannot be palpated, as it becomes retroperitoneal behind the descending part of the duodenum. To the right of the gall-bladder the hand will pass beneath the right lobe of the liver and above the transverse mesocolon and the upper pole of the right kidney, as far as the posterior abdominal wall (twelfth rib); and in some cases slightly upward on the posterior surface of the liver, before meeting with the inferior reflection of peritoneum which forms the posterior layer of the right coronary ligament. Close to the spinal column the ascending vena cava can be palpated. Passing the hand to the left of the gall-bladder, along the inferior surface of the left lobe of the liver, the fingers are arrested within 6 or 7 cm. by the attachment of the gastro-hepatic omentum along the transverse fissure of the liver. This fissure is limited on the right by the neck of the gall-bladder and the cystic duct, and on the left by the round ligament within the folds of the falciform ligament attached to the longitudinal fissure of the liver (Fig. 9). By now passing the hand further to the left, the left extremity of the gastro-hepatic omentum is reached, enclosing the esophagus, and the hand can be pushed backward between the cardiac end of the stomach below and the left lobe of the liver above until the posterior layer of the left lateral ligament is encountered, at the posterior surface

of the left lobe of the liver (Fig. 15). The Spigelian lobe may be palpated by passing the finger through the foramen of Winslow, and then upward between the spinal column (tenth and eleventh dorsal vertebræ covered by the diaphragm) and the liver. The surface of the liver so reached is the Spigelian lobe (Fig. 13). It is wholly within the lesser peritoneal sac. Its right boundary is formed by the inferior vena cava, its left by the esophagus and cardia of the stomach, its upper boundary by the coronary ligament of the liver, and its lower by the transverse fissure of the liver (attachment of the gastro-hepatic omentum). As the finger lies in the foramen of Winslow that portion of the liver immediately

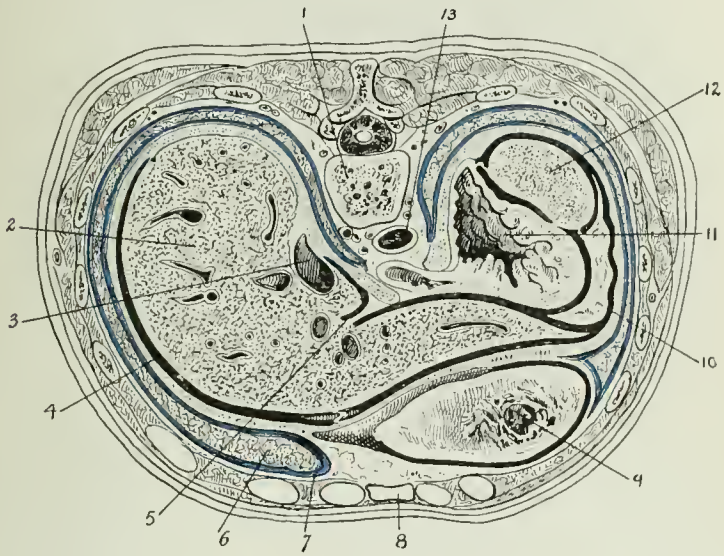

FIG. 13.—Diagram of Peritoneal Reflections in a Horizontal Transverse Section Passing Through the Pylorus.

1. Body of eleventh thoracic vertebra.
2. Liver.
3. Inferior vena cava.
4. Peritoneal cavity (black) between right lobe of liver and diaphragm.
5. Peritoneum covering Spigelian lobe of liver.
6. Margin of right lung.
7. Pleural cavity (blue).
8. Ensiform process of sternum.
9. Heart.
10. Great Omentum.
11. Stomach.
12. Spleen.
13. Aorta.

above it is the caudate lobe, connecting the Spigelian to the right lobe; the hepatic artery lies below the finger, being on its way from the celiac axis to the gastro-hepatic omentum; and the vena cava lies between the finger and the vertebral column.

The gall-bladder, which has already been mentioned, deserves further notice. Being formed as an outgrowth from the duodenum

along with the liver, it grows forward beneath this organ, and is enveloped in peritoneum except along its hepatic surface (Fig. 14). Sometimes a fold of peritoneum passes nearly directly downward from the fundus of the gall-bladder to the hepatic flexure of the colon (cystico-colic ligament), but more frequently the peritoneum covers the under surface of the gall-bladder closely, and passes thence to the duodenum. These peritoneal folds have been particularly studied by Sencert (1903).

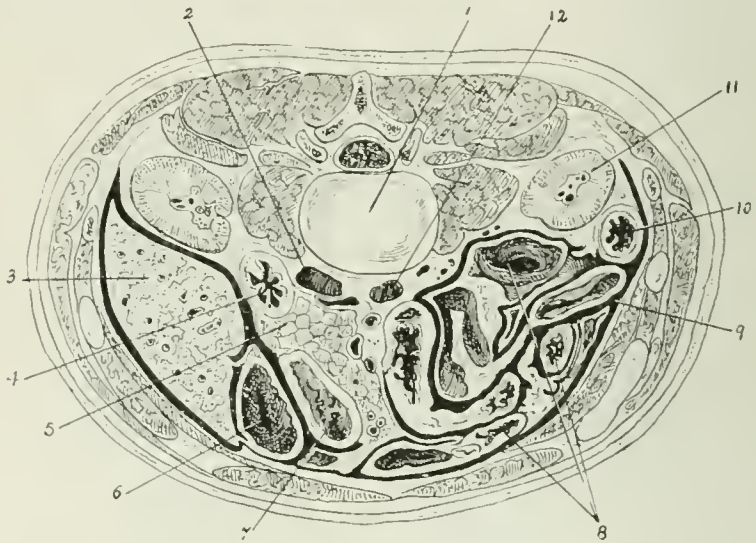

FIG. 14.—Diagram of Peritoneal Reflections in Transverse Horizontal Section Through the Head of the Pancreas.

1. Cartilage between second and third lumbar vertebræ.
2. Inferior vena cava.
3. Liver.
4. Ascending colon.
5. Head of pancreas.
6. Gall-bladder.
7. Descending duodenum.
8. Small intestines.
9. Peritoneal cavity.
10. Descending colon.
11. Left kidney.
12. Aorta.

The cystic duct is from 2.5 to 6.5 cm. in length, and joins the hepatic duct at an acute angle, to form the common bile duct. The cystic duct is about 2.5 mm. in diameter. The hepatic duct is usually only 2.5 to 4 cm. in length, and is formed by the coalescence of the right and left bile ducts descending from the liver. Its diameter is 4 to 6 mm. The common bile duct is from 2.5 to 7 cm. or more in length, compensating for the shortness of the other ducts when they are of less than average length. It is about 6 mm. in diameter. It ordinarily commences a little above the upper level of the pylorus, and passes down

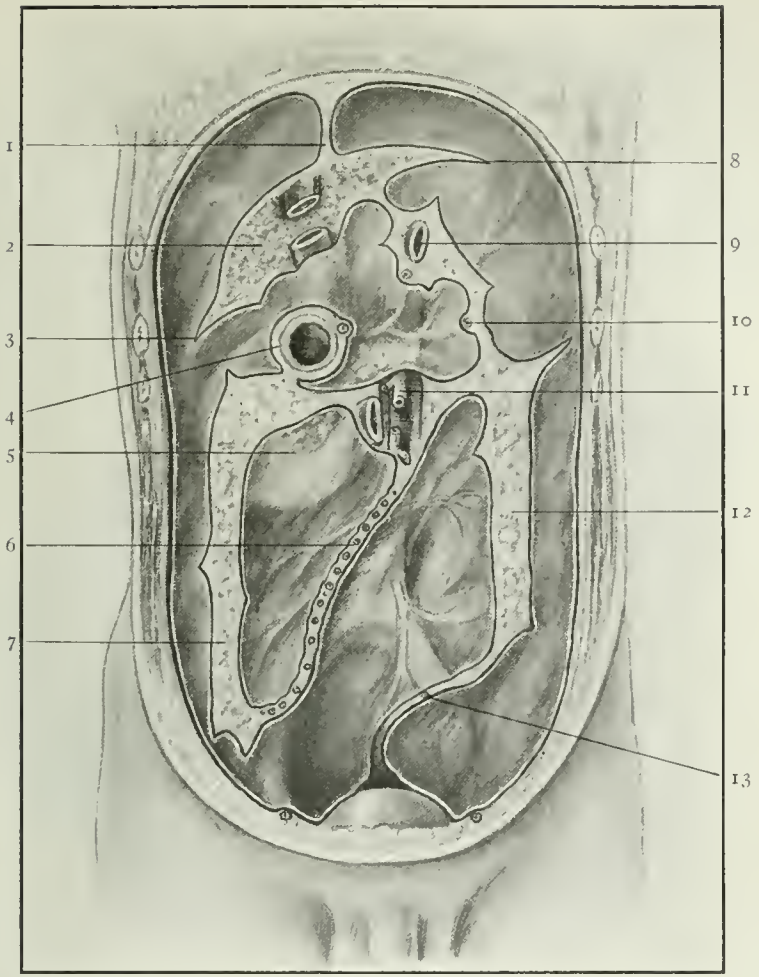

FIG. 15.—Diagram of Peritoneal Reflections after Removal of all Viscera.

1. Suspensory of falciform ligament of liver.
2. Extraperitoneal area between layers of right coronary ligament of liver, with vena cava.
3. Right lateral ligament of liver.
4. First part of duodenum with hepatic artery. Between duodenum and right coronary ligament of liver, is the foramen of Winslow.
5. Retroperitoneal course of duodenum surrounding head of pancreas.
6. Mesentery of jejuno-ileum.
7. Ascending meso-colon.
8. Left lateral ligament of liver.
9. Cardiac orifice of stomach, with gastric or coronary artery just below it.
10. Splenic artery in the gastro-splenic omentum.
11. Superior mesenteric artery, sectioned; the duodenum passes between it and the aorta.
12. Descending meso-colon.
13. Meso-sigmoid

behind this and in front of the pancreas, until it is joined on its posterior side by the pancreatic duct (Fig. 16). As it passes downward it may be completely enclosed in pancreatic tissue. This is the case in about two-thirds of the cases examined at autopsy. The combined pancreatic and bile ducts then traverse the postero-internal duodenal wall obliquely for about 2 cm. and empty into the interior of the descending duodenum about 7 to 10 cm. beyond the pylorus. To obtain a good view of this opening it is necessary to open the anterior duodenal wall, when the orifice of these ducts will be perceived as a slight pro-

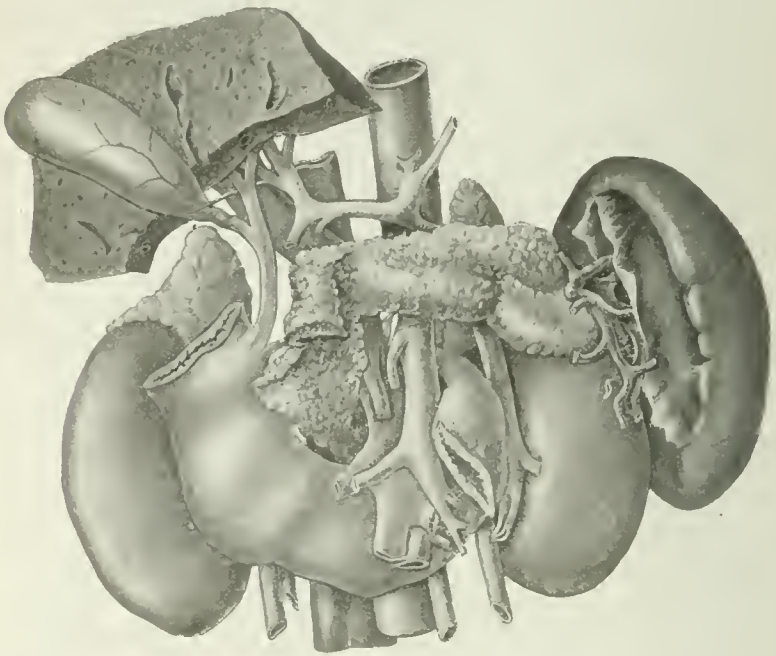

Fig. 16.—The Bile Ducts and Their Relation to the Duodenum and Pancreas.

jection of the mucous membrane (papilla of Vater) guarded on its superior surface by a further fold or hood of mucous membrane, which is provided with a frenum (Fig. 17). Other similar folds are sometimes found surrounding the papilla of Vater on all sides. Gall stones not infrequently lodge in the dilated portion of the duct known as the ampulla of Vater, just outside the duodenal opening.

The *arterial supply* of the liver is conveyed to it almost entirely through the hepatic artery, a branch of the celiac axis. By a somewhat semicircular course, with the convexity forward, this artery passes across the inferior border of the foramen of Winslow to reach the upper border of the pylorus, where it enters the gastro-hepatic

omentum, holding here a position to the left and in front of the portal vein, which lies behind and between the hepatic artery and the bile duct. Its length is from 4 to 5 cm. and in diameter it is not far from 6 mm. On reaching the transverse fissure of the liver or before, the hepatic artery divides into two or more branches: of these, one passes obliquely to the right, usually behind, but occasionally in front of, the bile ducts, and gives off the cystic artery to the gall-bladder (Fig. 16). The cystic artery lies between the cystic and hepatic ducts crossing sometimes in front, sometimes behind the hepatic or common duct, and

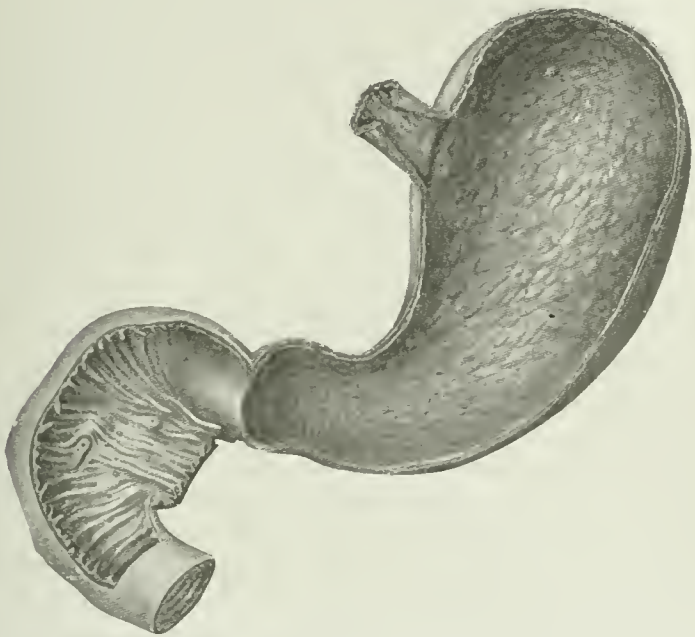

FIG. 17.—Interior of Stomach, Pylorus, and Duodenum, Showing Papilla of Vater, also Orifice of Duct of Santorini.

on reaching the neck of the gall-bladder divides into a superior and an inferior branch which supply the corresponding surfaces of the gall-bladder. The left hepatic artery, which is shorter than the right, passes to the left extremity of the transverse fissure, and supplies branches to the Spigelian and left lobes of the liver. The number and course of these terminal branches of the hepatic artery is exceedingly variable.

The *veins* of the liver collect the blood within its lobules, and, by radicles of gradually increasing size, finally empty by two or three trunks directly from its posterior surface into the inferior vena cava. Except for semilunar folds at the entrance of these veins into the vena

cava, no valves exist throughout the hepatic veins. The blood is urged onward largely by the alternate contraction and expansion of the liver which occurs during respiration.

The *portal vein*, as is well known, is formed in front of the body of the first lumbar vertebra by the junction of the superior mesenteric and splenic veins. At its origin it lies between the head of the pancreas in front and the inferior vena cava behind. Then passing behind the pylorus and first part of the duodenum, it enters the folds of the gastro-hepatic omentum, lying behind and between the hepatic artery on the left and the bile duct on the right. In the connective tissue which surrounds it lie numerous filaments of the hepatic plexus of the sympathetic nerve, as well as some efferent lymphatics from the liver. On reaching the transverse fissure, the portal vein divides into two branches, right and left, distributed to the corresponding lobes of the liver. The main trunk is about 7 to 10 cm. in length. The portal system of veins drains the stomach, the whole of the small intestine, the vermiform appendix, the cecum, the ascending, the transverse, and most of the descending colon, as well as the spleen and the pancreas. The cystic vein of the gall-bladder also empties into the portal vein.

There are certain definite connections between the portal vein and the systemic veins, which are of importance in various hepatic conditions. These connections may be classified as: (1) those within the falciform ligament of the liver, namely, a small vein which sometimes is present, as the remains of the umbilical vein, and other small veins, known as para-umbilical veins, which surround the round ligament of the liver and anastomose with the epigastric and mammary veins of the abdominal wall; (2) anastomoses between various radicles of the portal system and veins of the posterior abdominal wall—as between those of the pancreas, of the duodenum, and of the ascending colon, with veins of the posterior abdominal wall, such as the lumbar veins, the azygos veins, etc.; (3) between the superior hemorrhoidal veins (tributaries of the portal) and the middle and inferior hemorrhoidal veins (tributaries of the internal iliac veins); (4) certain anastomoses between the tributaries of the coronary veins and the esophageal veins; and (5) between the veins of the portal system and the phrenics, at the uncovered area of the liver. Of these various communications the most important are the first, third and fourth classes, especially the third and fourth. Portal obstruction may produce the "caput Medusæ" around the umbilicus in affecting the veins of the falciform ligament; in this case the current of blood flows away from the umbilicus and the para-umbilical veins. But if the "caput Medusæ" is due to

obstruction of the inferior vena cava, then the course of the blood is reversed, and it drains toward the navel into the veins of the round ligament. In such cases there is also sometimes enlargement of a superficial vein connecting the epigastric or external iliac vein with the axillary, which is easily detected as it runs up the side of the abdomen and chest. Enlargement of veins in the second classification is seen chiefly where the pancreas, duodenum, etc., are bound down by adhesions, and their normal drainage into the portal system is interfered with. Hemorrhoids, one of the most annoying and constant symptoms of portal obstruction, are produced by overdistention of the superior rectal veins; and as the communication between them and the middle and inferior hemorrhoidal veins is free, all three sets of rectal veins are frequently found to be varicose. The importance of varicose veins of the esophagus as a symptom of portal obstruction is now well recognized, and we have knowledge of more than one patient who has bled to death from the rupture of unsuspected varicose esophageal veins.

The *lymphatics* of the liver are divided into internal and external. The former accompany the branches of the hepatic and portal veins, and are not of so great surgical importance as the external set. Those accompanying the hepatic veins empty into the lymph nodes situated on the upper surface of the diaphragm just above the caval opening; while the lymph vessels accompanying the portal veins empty into the nodes about the neck of the gall-bladder and the cystic duct. The external lymphatics of the liver lie under its peritoneal covering, and in the connective tissue of the capsule of Glisson, and all drain away from the interior of the liver. They consist of several groups: (1) Those on the upper or convex surface: (a) Three or four branches pass forward along the upper surface of the liver into the falciform ligament, where they unite into a single trunk which enters the chest through the small diaphragmatic opening at the side of the xiphoid cartilage, and joins the anterior mediastinal nodes, eventually emptying into the right lymphatic duct. (b) A similar group turns downward over the anterior border of the liver to its under surface, passes along the longitudinal fissure to the transverse fissure, and thence to the nodes of the gastro-hepatic omentum. (c) Some lymphatics from the superior surface of the right and left lobes of the liver pass to the right and left lateral ligaments, and enter the anterior mediastinal nodes or the lower end of the thoracic duct. (2) The external lymphatics from the under surface of the liver may be classified as follows: (a) Those on the right of the gall-bladder empty into the lumbar nodes; (b) those on the left of the gall-bladder pass to the esophageal nodes and to the nodes along

the lesser curvature of the stomach; and (c) those surrounding the gall-bladder form a plexus and pass to the nodes of the gastro-hepatic omentum. It is thus seen that the lymph nodes around the neck of the gall-bladder and in the gastro-hepatic omentum drain the following areas of the liver: anterior median portion of the convex surface, gall-bladder area, and all of the inferior surface of the left lobe; as well as receive the deep lymphatics which run with the branches of the portal vein. These nodes, therefore, in the gastro-hepatic omentum are the most important surgically of all the lymph nodes in connection with the liver, and are frequently found enlarged in gall-bladder diseases, in malignant growths, and in inflammations of the liver, as well as in hepatic cirrhosis. They are also sometimes enlarged in Hodgkin's disease, and by compression of the portal vein have been said to cause ascites in this affection; but this latter action has been questioned. As Rolleston (1905) has pointed out, neoplasms may work their way into the liver by the portal fissure against the lymph stream which normally flows outward in this location. It is well recognized that lymph currents are very easily reversed by obstruction to their normal course.

The *nerve supply* of the liver has recently been studied by Latarjet, Bonnet and Bonniot (1920): they recognize a main gastro-hepatic nerve, which runs in the gastro-hepatic omentum from the left (anterior) pneumogastric nerve and plexus to the liver. The main sympathetic nerves arise in the celiac plexus: from the semilunar ganglion the *posterior hepatic plexus* travels across the hepatic artery and along the left side of the choledochus and hepaticus to the liver; while the *anterior hepatic plexus* travels along the right side of the choledochus to the liver. The right phrenic nerve is also connected with the hepatic nerve; it is derived mainly from the fourth cervical nerve, which also sends a branch—the supra-acromial nerve—to the integument of the point of the shoulder, thus explaining the "shoulder-tip pains" encountered in certain hepatic affections.

The disposition of the peritoneum and the relations of neighboring organs to the liver form what was well described by M. H. Richardson as the "liver pouch." This is of vast importance in preventing infection of the general peritoneal cavity in diseases of the gall-bladder and other organs in the upper right abdominal quadrant. Limited by the under surface of the right lobe of the liver above, by the duodenum and spinal column toward the median line, and by the transverse mesocolon below, this pouch readily collects all extravasated fluids and becomes a valuable site for drainage, which may in some instances

be most readily procured by an incision into its floor from the loin, below the tip of the twelfth rib.

Duodenum.—The duodenum, with the exception of its first portion, is entirely retroperitoneal, and is covered in front by so many important structures that only its first and second portions are readily accessible during life. The first portion, continuous with the pylorus, is easily reached above the transverse colon, and to the left of the gall-ducts. The descending portion is best exposed by dividing the outer layer of the ascending mesocolon throughout its upper third, when by rolling the hepatic flexure of the colon together with the pylorus toward the patient's left, the outer and posterior walls of this portion of the duodenum will come into view; the bile and pancreatic ducts are thus accessible to surgical treatment (Fig. 163). To expose the transverse portion of the duodenum the least dangerous plan is to divide the inferior layer of the mesentery of the small intestine, just above the bifurcation of the aorta; this gives access to the duodenum as it crosses the spinal column immediately below the superior mesenteric vessels. In Jaboulay's entero-anastomosis (1902) the transverse portion of the duodenum on the right of the superior mesenteric artery was utilized. The duodeno-jejunal flexure is readily found by turning the transverse colon upward and seeking for the origin of the jejunum as the small intestine emerges from beneath the transverse mesocolon (Fig. 18). Below and to the left of the terminal portion of the duodenum, with its orifice directed upward, is the duodeno-jejunal fossa. It is found in about 48 per cent. of cases, and may be the seat of retroperitoneal hernia.

The relations of the duodenum to surrounding organs have already been considered. Of these the most important are its relations with the gall-bladder and with the transverse colon. From the former it is separated by two serous surfaces, the visceral layer covering its first and second portions, and the visceral layer of the gall-bladder itself (Fig. 14). Adhesions are frequent, and ulceration of the duodenum may extend into the gall-bladder, or *vice versa*. From the transverse colon the descending duodenum is separated only by a little areolar tissue, there being no peritoneum between the two organs where the root of the transverse mesocolon crosses the duodenum. The duodenum is fixed in its position not only by its retroperitoneal situation, but by peritoneal reflections to the liver and gall-bladder from its initial portion, and by the ligament of Treitz from the duodeno-jejunal juncture to the diaphragm. Besides these means of fixation, there are the insertion of the bile and pancreatic ducts, and the

proximity of the superior mesenteric vessels in front and above the duodenum.

Ochsner described (1906) a sphincter of the duodenum, consisting

FIG. 18.—The Origin of the Jejunum, and the Duodeno-jejunal Fossa, Exposed by Turning the Transverse Colon Upward. The Jejuno-ileum has Been Drawn Far to the Right.

of a more or less well defined band of thickened circular fibres, usually some distance below the entrance of the bile and pancreatic ducts. He thought it of assistance in the phase of duodenal digestion by retain-

ing the chyme in the duodenum until it was ready to be discharged into the jejunum. Boothby (1907) was unable to confirm the existence of such a sphincter in any of 25 specimens examined at autopsy.

Pancreas.—The pancreas, which is also retroperitoneal, is covered anteriorly by the posterior parietal layer of peritoneum which forms the lesser peritoneal cavity. It is best exposed through the gastrocolic omentum (Fig. 159), or by passing beneath the great omentum and detaching this from the transverse colon (Fig. 11). Neither of these routes gives adequate exposure of the head of the pancreas. The posterior portion of the head however, may be partially exposed by mobilization of the duodenum (Fig. 163). But by detaching the great omentum from the transverse colon, and then dividing, within

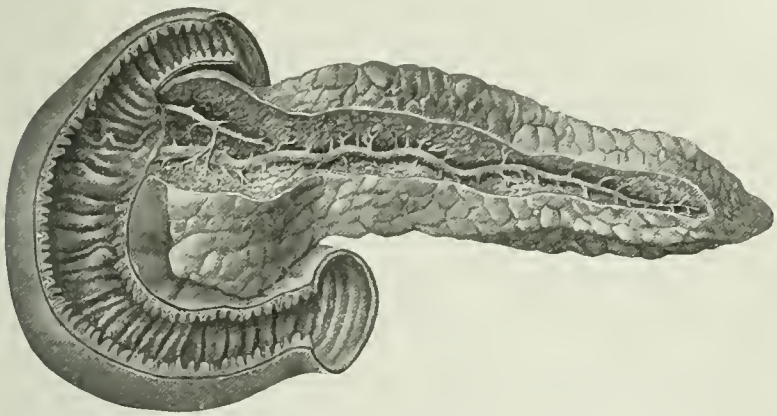

FIG. 19.—The Pancreas, its Ducts, and Their Relation to the Duodenum.

the lesser peritoneal cavity, the superior layer of the transverse mesocolon over the pancreas, a fairly free exposure of this organ throughout its whole length is obtained.

The *bloody supply* of the pancreas is derived chiefly from the pancreatic branches of the splenic. This artery runs in a very tortuous course along the upper border of the pancreas. The pancreas being situated just between the celiac axis above and the superior mesenteric vessels below, injuries of this organ are frequently fatal from hemorrhage if not from sepsis or interference with the digestive functions. The close relation borne by the pancreas to the posterior wall of the stomach explains the frequency with which this organ is involved in carcinoma of the stomach. Primary carcinoma may also affect the pancreas, and the growth, if it involves the head of the organ, may cause

occlusion of the common bile duct and consequent jaundice. If the malignant growth affects that part of the organ in relation with the aorta, it may simulate aortic aneurism by causing a swelling over which transmitted pulsation is detected.

The *lymphatics* of the pancreas are discussed in Chapter XXI in connection with infection of this organ.

The *nerves* of the pancreas are branches of the splenic plexus of the sympathetic.

CHAPTER II

PHYSIOLOGY OF DIGESTION

The region of the upper abdomen includes practically all of that portion of the intestinal tract, with its annexed glands, concerned in the process of digestion. Although the intestines below the duodenum serve as a receptacle for the ingested food, and although the succus entericus has certain important functions to perform upon the ingesta, yet these parts of the digestive tube are concerned more particularly in the processes of absorption and elimination than in that of digestion.

No less striking than the development of the surgery of the abdomen in recent years, are the valuable contributions to our knowledge of the physiology of digestion which are products of the same period. There is no better illustration of the fact that the solution of physiological problems, which has baffled master minds of bygone days, has been rendered possible only by the employment of modern surgical methods in investigations, than the fact that the observations of Beaumont upon Alexis St. Martin, who had a gastric fistula caused by a gunshot wound, have been considered authoritative from 1825 until recent times. The results of modern investigation have such important clinical relations to the surgery of the digestive system that they well merit consideration here.

Intricate though the processes of digestion may seem at first sight, a knowledge of embryology renders these problems simple.[1]

Perusal of the preceding chapter has revealed the fact that, in the fetus, the alimentary canal early assumes the form of a straight tube. A portion of this tube becomes dilated, and forms the stomach. New relations are established by rotation of the stomach and intestines. Evaginations of the wall of the duodenum create two essential glands, —the liver and the pancreas. Analysis of this growth-process impresses one at the outset with two striking features: first, the very early appearance of the rudiments of the liver and pancreas, and secondly, the great extent of intestinal coiling. The first feature suggests that

[1] As W. J. Mayo expresses it, man prepares his food with the organs which have their origin in the foregut, and absorbs his nutrition from the derivatives of the midgut; that is, he eats with the jejunum and the ileum and drinks with the cecum.

glandular activity is to be closely associated with the workings of the digestive tube. As a matter of fact, the latter is lined from the lips to the anus with countless glands. The development of any gland is very simple, but specialization occasions complexity. Cylindrical ingrowth of the epithelium (which in this case lines a cavity) into underlying tissue, with subsequent hollowing-out of the cylinders, constitutes a gland of the simple unbranched tubular type, such as is present in the fundus of the stomach, or, as Lieberkühn's glands, throughout the small and large intestines. Offshoots from the cylinders establish the type of simple branched tubular glands, represented by the countless small serous and mucous glands of the oral cavity, and by the glands at the pylorus of the stomach and in the duodenum (Brunner's glands). If the endings of the epithelial plugs expand and secondarily give rise to primary alveoli, there is produced the racemose type of glands, such as the salivary glands and pancreas. Further and more complicated modifications of structure produce a reticular tubular gland, such as the liver, in which instance the anastomosis between the branches of the tubular glands is so extensive that a net-like structure is produced. The salivary glands, pancreas, and liver are distinguished by ducts of various lengths, so that we may designate them extra-mural glands. To sum up, therefore, we are impressed with the fact that innumerable glands line the digestive tube, and that, where the requirements of the organism demand it, several glands are further developed and specialized, and are removed from the walls of the digestive tube to undergo further enlargement, but are still connected with this tube by ducts of varying lengths. The aggregate of output of these glands is, therefore, very considerable. Sappey has calculated that the surface of the mucous membrane of the human stomach presents over 5,000,000 orifices of gastric glands.

Digestion may be defined as a mechanical and chemical process by which food is prepared for absorption. Of course it is useless, in a work which aims to treat of practical questions in the surgery of the upper abdomen, to discuss at length the various theories and facts involved in the study of human digestion; but it is unavoidable to offer readers of such a work some sort of reference chapter, which will serve to illustrate the main principles of physiology involved, and will enable them to apply their knowledge when discussing the pathology and treatment.

Digestion means the intake of food, its mastication, its exposure to the action of various secretions, to intestinal movements, to absorption and finally, if of no further use to the organism, its elimination. Na-

ture prepares a very difficult gauntlet for the food to run, so as to extract from it every possible iota of nutrition. Prolonged contact with the food is secured, in the first instance, by coiling of the small bowel, and sacculation of the large; and, secondarily, by the presence of the valvulæ conniventes and villi in the small intestine. Not only is the area of absorption incalculably increased in this way, but also the capacity of secretion is greatly augmented.

As regards function, practically all of the glands in connection with the gastro-intestinal tract are capable of secreting mucus, the lubricant. Some few secrete serous fluid, the diluent. To fewer still is reserved the property of secreting more highly specialized products, the enzymes. Two, the liver and pancreas, are concerned also in internal secretion.

Food stuffs, as we all know, are classed as Proteids, Carbohydrates, and Fats, respectively represented by meats, by sugars and starches (rice, macaroni, bread, etc.) and by fatty substances such as butter, eggs, cheese, and fat of meats. When digested and therefore prepared for absorption, it may be stated with sufficient accuracy that proteids become peptones, that carbohydrates become maltoses, and that fats are absorbed practically unchanged.

The proteid is acted upon by three agencies, namely, the pepsin of the stomach, the trypsin of the pancreas, and the bacteria in the large intestine. The carbohydrate constituent is disposed of by three agencies, the ptyalin of the parotid glands, the amylopsin of the pancreas, and amylolytic enzymes from Lieberkühn's gland of the small intestine. The fats are attacked by the lipase (steapsin) of the pancreas, an enzyme the activity of which is enhanced by the bile. There are other less important enzymes.

The mechanical part of digestion is, or should be, performed largely by the cooking and by the mastication of the food. After being swallowed, the action of the stomach consists both in a churning movement which mixes the bolus of food with the gastric juices, as well as in a rhythmic peristalsis by which the food is from time to time urged forward into the duodenum. Beyond the pylorus, practically the only mechanical action to which the ingesta are subjected, apart from the peristaltic motion of the intestines, is represented by the dilution which they undergo by admixture with the bile and with the pancreatic and intestinal juices. So great is this dilution that although much of the fluid portion of the food is absorbed while passing through the small intestine, yet even when the cecum is reached the intestinal contents are always very soft, and usually semifluid.

From the standpoint of anthropology, cooking of foods is not essen-

tial to the welfare of mankind, but is a product of civilization. This trespass upon Nature has resulted in an inherited tendency of the human teeth toward premature decay on account of decreased necessity for the grinding action. The possession of poor teeth predisposes to gastro-intestinal affections, whilst a good set acts as a powerful prophylactic. However, cooking of meat sets free the muscle fibres by its action on the connective tissue, which it partly transforms into gelatin. Cooking of vegetables springs the pellicle and renders the starch more soluble. In bread baking, the dough is spongified by the carbonic acid formed from the yeast. Furthermore, cooking kills parasitic ova, and renders food sterile.

The **prephase of digestion** is initiated by the senses of sight, smell, and hearing, and also by the thought of, and longing for, food. Careful and attractive preparation of food, and savory odors that emanate from it, powerfully whet the appetite. Appetite may be stimulated through the sense of hearing, by clatter of dishes and the sizzling of meat directly removed from the fire. The craving for food is instinctive, and is the basis of the appetite.

Salivary Digestion.—These psychic events bring about a flow of saliva, as a preparatory step to the introduction of food into the mouth. The presence of food in the mouth causes additional flow of saliva, which now is adapted to the character of the material ingested. The secretion from the parotid glands is serous, and contains the enzyme, ptyalin; that from the sublingual and numerous minute glands in the mouth is mucous, whilst the secretion of the submaxillary glands is mixed, serous and mucous. By moistening the food, saliva aids mastication, and by enveloping the hard and bulky bolus with mucin, it facilitates deglutition. It dissolves the soluble, a step necessary for inauguration of taste sensations. Furthermore, saliva is protective in that it tests materials introduced into the mouth, neutralizing deleterious properties, rejecting the harmful, and washing out injurious substances which might enter the blood through contact with the mucous membrane. A specific excitability is manifested by the salivary glands, since fresh, moist food creates but little secretion, whilst dry materials induce a copious flow. The amylase (ptyalin) from the parotid gland changes starch into dextrin and maltose. Owing to the short stay of food in the mouth, salivary digestion takes place chiefly in the stomach. Although destroyed by gastric juice, yet ptyalin continues its action in the interior of the bolus of food until the gastric juice has completely penetrated the mass, a process which requires from 20 to 40 minutes.

Gastric Digestion.—Before the food reaches the stomach, gastric juice has been secreted by the innumerable tubular glands which stud thickly the gastric mucosa. The excitant of this preliminary flow is psychic; in fact it resides in the appetite, and hence we may speak of "appetite-juice." The latter, which appears within 5 minutes, is copious in amount, and strong in digestive power. A good appetite in eating is equivalent from the outset to a vigorous secretion of the strongest juice; lacking appetite, this juice is also absent. Restoration of appetite means gastric juice in plenty, wherewith to inaugurate digestion.

The qualitative secretion of the gastric glands varies in different parts of the stomach. In the fundic region pepsin, rennin, and scanty amounts of hydrochloric acid are secreted; in the pre-pyloric region the same substances and most of the hydrochloric acid, the latter fact being indicated by the deeper red color of the mucous membrane in this area; whilst in the pyloric region pepsin and rennin only are produced.

In the stomach the ingesta undergo equalization of temperature, maceration by the gastric juice, and conversion into chyme. Although ptyalin is destroyed in an acid medium, yet salivary digestion of carbohydrates may proceed for 30 minutes in the stomach, not only on account of the slight acidity of the gastric juice in the fundus where the food first lodges, but also because of the length of time required for complete penetration of each bolus of food by the gastric juice. The copious amount of "appetite-juice" is now augmented by a second quantity of juice, produced chemically. The first complement of juice decreases in amount as the second increases. The latter depends on the production in the pyloric mucous membrane of a specific substance or hormone, which acts as a chemical messenger to all parts of the stomach, being absorbed into the blood and thence exciting the activity of the various secreting cells in the gastric glands.

Just as the acidity of the gastric juice is detrimental to the action of ptyalin, so is it essential for the action of the ferment, pepsin. During the time usually occupied by gastric digestion, namely, from 3 to 6 hours, proteids are prepared by the pepsin-hydrochloric acid for subsequent digestion by the enzyme, trypsin, in the small intestine. With this end in view, most of the proteid is converted in the stomach into its first products of hydration, namely, peptones and proteoses, in which state the proteids of the food are normally passed on into the duodenum, having been rendered more amenable to the action of trypsin. Therefore, disposal of proteids occurs in the cycle of peptic-tryptic digestion.

Rennin possesses the specific action of curdling milk, which is brought about by the coagulation of caseinogen.

Fat undergoes no digestive change in the stomach. It is merely liquefied by the bodily heat, dissociated from other foods by the specific proteolytic action of the pepsin-hydrochloric acid, and mixed with the chyme in the form of a coarse emulsion. Fat inhibits the work of the gastric glands, both from a quantitative and from a qualitative point of view: hence the omission of fat other than in emulsion, from a corrective dietary. On the contrary, water and extracts of meat exert a stimulating effect upon the secretion of gastric juice.

Absorption from the stomach is very slight, although alcohol is absorbed readily, and certain soluble drugs may be.

Gastric digestion continues until the whole of the stomach contents is discharged from the pylorus as the semifluid chyme. After this event, the stomach enters upon a resting stage, during which its cavity is nearly obliterated.

Intestinal Digestion.—The fluid chyme, on entering the duodenum, is subject at once to the influence of the secretions of three different sets of glands, namely: (1) The intestinal glands, including those characteristic of the duodenum, called Brunner's glands; (2) the pancreas; (3) the liver. The ducts of the two latter in man have a common opening into the duodenum, and there is a co-operation between all three juices for the production of the intestinal digestive fluid.

The flow of pancreatic juice is initiated chemically. The epithelial cells lining the intestines contain a body—pro-secretin—which, under the influence of agents such as acids, undergoes hydrolysis with the splitting off of a new body, termed secretin. The latter, on absorption into the blood, acts as the chemical messenger (hormone) to the pancreatic cells. Sleep does not hinder pancreatic secretion.

The pancreatic juice is alkaline, a reaction that corresponds closely in degree to the acidity of the gastric juice. Aided by the bile and alkaline juice from the intestinal glands, the pancreatic juice neutralizes the acid chyme, with the result that a neutral fluid, in which the processes of intestinal digestion will continue is produced in the duodenum.

Secretion of pancreatic juice starts shortly after entrance of food into the stomach, and rapidly reaches a maximum in from 2 to 4 hours, whilst by the seventh hour it has practically ceased. The character of the food modifies the composition of the secretion. The pancreatic juice when it reaches the duodenum contains three enzymes,

of which trypsin is proteolytic; amylopsin, amylolytic; and steapsin, lipolytic. Secretion evoked by proteids abounds in trypsin; that by carbohydrates, in amylopsin; and that by fats, in steapsin.

If trypsin be proteolytic, the question would naturally arise, why should not this enzyme digest the intestinal mucosa? The answer is that trypsin is not a secretion, but a resultant. The secretion is trypsinogen, a pro-enzyme, which is converted into trypsin by enterokinase, a product of the duodenum and jejunum. Therefore, until enterokinase trans-substantiates trypsinogen into trypsin by catalysis, no proteolysis is manifested.

Trypsin continues the transformation of proteids which was begun in the stomach. It also completes the cycle of peptic-tryptic digestion. Trypsin, however, acts more rapidly and powerfully than pepsin, and breaks up the proteid molecule more completely. Thus, the peptones and proteoses, prepared from proteids by the pepsin, and delivered by the stomach into the duodenum, are further split by trypsin into amido-acids.

Amylopsin acts upon starches in very much the same way as does ptyalin. The carbohydrates that have escaped the action of ptyalin are hydrolyzed in the duodenum, by amylopsin, into maltose and dextrin; and these, in turn, are converted into dextrose by the maltase of the succus entericus.

Steapsin, materially aided by bile, splits up neutral fats into glycerin and free fatty acids.

The Secretion of Bile.—Since bile reaches the duodenum through an orifice common to it and to the pancreatic juice, the natural inference is that these two fluids co-operate in their action, and that bile is of direct use in digestion. As a matter of fact, bile increases the action of steapsin two to three fold, and that of trypsin and amylopsin about two fold. Indeed, bile is of great value in digestion, and plays an important *rôle* in this process. Beginning almost immediately after taking food, the secretion of bile attains its maximum with the pancreatic juice in the third hour, is regulated by the same laws that govern the flow of other digestive juices, and then rapidly declines. Thus, bile is produced by the same agent as pancreatic juice, namely, by secretin. Furthermore, bile flows as long as digestion lasts, but with definite fluctuations in quantity and quality, dependent upon the nature of the food.

The bile is being constantly formed in the liver, and, during the intervals of digestion, is stored up in the gall-bladder. Its pressure is not known accurately, but is believed to be always greater than that of

the blood in the portal vein. In amount the bile varies from twenty to twenty-seven ounces (600 to 800 cc.) daily. Its descent from the liver is probably aided by the changes in the bulk of the liver produced by respiration, as well as by the *vis a tergo* of the more freshly formed bile. The contractions of the gall-bladder which are usually believed to occur, are said to be produced by sensory stimulation of the mucous membrane of the stomach or duodenum through reflex nervous action. The presence of chyme in the duodenum causes, by means of secretin, a contraction of the gall-bladder and a relaxation of the sphincter muscle of Oddi surrounding the duodenal orifice of the bile duct, with consequent ejection of bile. There is so little muscular tissue in the gall-bladder, however, that it is doubtful whether the gall-bladder does more than equalize the pressure in the bile ducts, like the air chamber of an hydraulic ram. It is an interesting fact that even though almost the whole amount of bile excreted be diverted through a biliary fistula, so that scarcely any of it reaches the intestinal tract, yet nevertheless the individual so affected may continue to enjoy good health, showing conclusively that bile is much more of an excretion than a secretion.

To sum up, the chief duty of the bile is to facilitate the transition from gastric to intestinal digestion, since it enters the duodenum at a spot where the acid peptic digestion gives place to alkaline pancreatic digestion; it arrests the action of pepsin, which is harmful to the enzymes of the pancreatic juice, and reenforces the enzymes of the latter, particularly by serving as a vehicle for the suspension and solution of the interacting fats, fatty acids, and steapsin.

Not only is bile important in digestion, but further, as an excretion, it is the channel by which the disintegration-products of hemoglobin are cast out from the organism.

Succus Entericus increases the activity of the pancreatic enzymes. Just as bile aids particularly the action of the pancreatic lipolytic enzyme, so does succus entericus augment the proteolytic. Hence both of these secretions are adjuvants of the pancreatic juice. Secretin is also the producer of succus entericus.

Succus entericus is a secretory product of some of the glands of Lieberkühn. Collectively, these glands, as well as those of the stomach, may be considered as an enzyme-producing entity which, instead of being gathered together to form an extramural organ, such as the liver or pancreas, is distributed throughout the intestinal wall, thence to discharge secretion directly into the lumen of the intestine.

Succus entericus, distinctly alkaline in reaction owing to sodium

carbonate, contains four or five enzymes that complete the digestion of food-stuffs begun in the stomach and duodenum, thus exercising a most important influence upon intestinal digestion. Of these enzymes two, enterokinase and erepsin, are concerned in proteolysis. Enterokinase, as has been seen, activates the proteolytic enzyme of the pancreatic juice, by converting the trypsinogen into trypsin. Erepsin supplements the work begun by trypsin, in that it causes further hydrolysis of peptones and proteoses.

Secretin, which initiates the secretion of bile, pancreatic juice, and succus entericus, is not an enzyme, but a definite chemical substance produced in the intestinal wall in a preliminary form, pro-secretin, which, influenced by acids, is converted to secretin. The latter is absorbed and carried to the glands, the secretion of which it evokes.

The remaining enzymes of succus entericus are concerned in the digestion of carbohydrates. They are maltase, invertase, and lactase, the last being present in young individuals and in those fed throughout life exclusively upon a milk diet. Maltase acts upon the products of the digestion of starches, namely, maltose and dextrin, converting them into dextrose. Invertase transforms cane-sugar into dextrose and levulose, whilst lactase changes milk-sugar into dextrose and galactose.

Absorption in the Intestines. In consequence of all these changes, the three classes of food-stuffs are reduced to a soluble condition, and in solution are taken up by the cells lining the intestine. The products formed in digestion largely disappear between the duodenum and the ileocecal valve. Carbohydrates are absorbed chiefly as simple sugars—monosaccharids. As dextrose, then, the sugars pass directly into the blood stream, by which they are distributed first to the liver and then to other organs of the body. In the liver the excess of sugar is removed from the blood and stored as glycogen. Alimentary glycosuria is a phenomenon arising from ingestion of larger amounts of carbohydrates than the liver can store up as glycogen, the excess being removed from the blood by the kidneys, and excreted in the urine. Any carbohydrates which escape absorption as sugar are apt to undergo acid fermentation from the action of the bacteria constantly present in the intestine.

Proteids, hydrolyzed during digestion into peptones and proteoses, or amido-acids, probably are absorbed as such, passing directly into the blood-vessels of the intestinal villi, and thence into the blood stream.

Fats are absorbed by the epithelial cells in the forms of fatty acids and glycerin, which, in turn, are immediately re-synthesized into in-

soluble neutral fats in the cells themselves. In the state of the fine emulsion—chyle—most of the fats reach the blood stream through the lacteals and thoracic duct. Absorption of split fats is considerably aided by the bile. Some of the fat reaches the liver by way of the blood stream, and undergoes accumulation in that organ. If an excess of fat were ingested, of if the flow of bile were decreased or stopped, a large percentage of fat would escape absorption and appear in the feces.

The secretion of the large intestine is alkaline, and contains much mucus, but is itself devoid of enzymes, those that are present having been contributed and passed along by the small gut. Since absorption and digestion are not completed in the small intestine, they are continued in the large. Furthermore the latter absorbs large quantities of water.

Bacterial Action.—The bacteria normally found in the intestinal tract are of considerable practical importance. At birth the digestive tract is sterile, but as soon as unsterilized food is ingested there are found bacteria of various kinds, pre-eminently the colon bacillus. As has been shown by numerous observers, the emptier the intestinal tract is of food, the fewer will be the bacteria present. In the stomach, bacteria disappear with the food, and when the stomach has been entirely empty for some time, its cavity is nearly sterile, owing to the antiseptic properties of the gastric juice. According to the investigations of MacNeal and Chace (1913) the fasting duodenum is almost free from living micro-organisms. Certain bacteria, especially Bacillus subtilis and Proteus vulgaris, are believed to have a proteolytic action, and thus to aid the peptic digestion. The duodenum in its upper part is singularly free from bacteria, probably due to the acid gastric juice; but as the small intestine is traversed, and the alkalinity of its contents increases, the bacterial content becomes greater and greater, reaching its maximum in the lower ileum; the bacteria are less numerous again in the colon.[1] The annexed diagram from Gilbert and Domenici describes these changes much more accurately than can mere words (Fig. 20). It is worthy of note that, just as purgation eliminates most of the bacteria with the intestinal contents so prolonged constipation and especially intestinal obstruction markedly increase the virulence of the intestinal bacteria.

In the small intestine, bacteria show activity by fermenting carbohydrates. This process exerts a restraining effect upon proteid

[1] The fact that injuries of the large bowel are more apt to be followed by peritonitis than are those of the small bowel probably may be explained by the greater virulence of the bacteria (especially the colon bacilli) which are here encountered.

putrefaction, which, on the contrary, is a constant and normal occurrence in the large intestine. In this way proteids that have escaped digestion and absorption are split up into various end-products, some of which are given off in the feces, whilst others are absorbed in part and excreted subsequently in the urine. The extent to which these bodies occur in the urine is an indication of the extent of putrefaction in the large intestine, a fact which possesses certain clinical value. Cellulose, for which there is no specific enzyme, is hydrolyzed by bacteria and thus rendered useful in nutrition. Aside from this, it may be said that bacterial fermentation is not essential for the welfare of the economy.

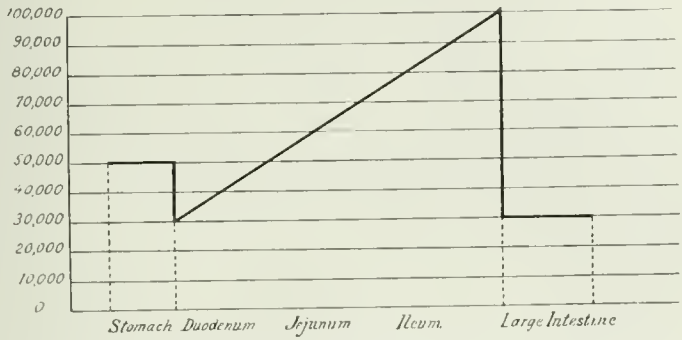

FIG. 20.—Graphic Representation of the Bacterial Content of the Gastro-intestinal Tract. (*Gilbert and Domenici.*)

Composition of the Feces.—The character of the food has an important influence upon the composition of the feces. Upon a diet composed exclusively of meats they are small in amount and dark in color; with an ordinary mixed diet the amount is increased; and it is largest with an exclusively vegetable diet, especially with vegetables containing a large amount of indigestible substances. Feces are made up of indigestible and undigested materials; products of intestinal secretions and of bacterial decomposition; cholesterin, excretin, mucus and epithelial cells, pigment, inorganic salts, and microorganisms. In addition, gas, arising from bacterial fermentation of proteids, is present in varying amounts.

The main function of the alimentary tract, therefore, is the presentation to the tissues of the body of the food-stuffs in a form in which they are directly assimilable.

Liver.—Our knowledge of the physiology of the liver, until recent years, has consisted in acquaintance with the facts that it produced bile, and that it served as a storehouse for carbohydrates, absorbed as

maltose and dextrose from the intestines through the portal system of veins, and stored up in the form of glycogen. This glycogen, by the metabolic action of the liver cells, may be again converted into maltose as occasion demands, and be given off into the general circulation for nutriment to the muscles and other structures of the body. Attempts,

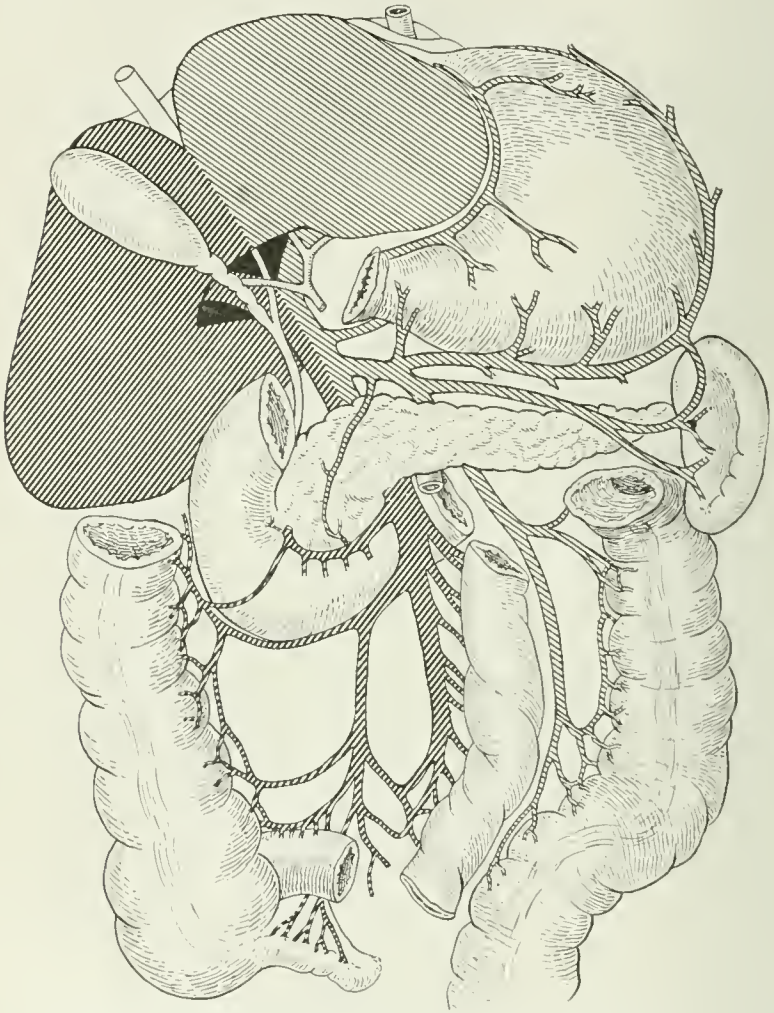

FIG. 21.—Diagram to Show the Relation of the Lobes of the Liver to Various Abdominal Structures, Indicated by Blood Currents in the Portal Vein.

however, have been made by Silvestri (1905) and others to specialize the functions of the right and left lobe of the liver. As has been known for some years, the right lobe of the liver is especially affected by enlargement in diabetes (supposedly a pancreatic disease); while in

Banti's disease, and other splenomegalies, the left lobe is more particularly affected. Silvestri records some experiments of his own, and refers to others by Glénard and by Sèrègé, which tend to confirm the idea that the left lobe of the liver is intimately connected with the stomach and the spleen, while the right has more definite relations with the pancreas and small intestines. Injections of staining fluids into the spleen invariably produce a discoloration in the liver limited to the left lobe; while similar injections into the superior mesenteric radicles, made very cautiously, so as not to disturb the venous current, always stained the right lobe of the liver much more than the left. It is to be hoped that some practical application may be made of the information thus gained.

The blood pressure in the portal vein and in the liver is very low, so that hemorrhage from the liver is readily controlled by pressure or by approximation of the lips of the wound in the liver by sutures.

The *internal secretions of the liver and pancreas* are so far too little understood for anything very definite to be said about their physiological activity. Their relations with the kinetic system, so graphically described by Crile, have not yet been shown to be of much practical interest.

One of the most interesting of the functions connected with the complemental metabolism of the liver, is its so-called detoxicating power over certain substances. In addition to its influence over certain alkaloidal and mineral poisons (such as nicotin, hyoscyamin, strychnin, quinin, atropin, morphin, antipyrin, peptone, and certain toxins) it is probable that the liver exercises a somewhat similar detoxicating power over certain forms of bacteria themselves. It is possible that incompetence of the spleen, which normally abstracts from the circulation pathogenic bacteria, may thus overwork the liver and in turn cause it to become incompetent. For the liver then will have to deal not only with bacteria arriving from the intestinal tract, but also with those which pass through the spleen. The state known as cholemia, formerly thought to be caused by suppression of bile, and therefore analogous to uremia, has been observed sufficiently often without obstruction to the flow of the bile to render it certain that it is caused by an auto-intoxication from changes in the liver substance, not due to the damming up of the biliary excretion. For although it is quite probable that this latter condition will in most instances so injure the liver cells as to be productive of cholemia, yet other factors such as biliary cirrhosis may be equally destructive to the liver, while the excretion of bile remains unimpaired.

The Movements of the Alimentary Tract.—Chemical digestion is supplemented by movements of the alimentary tract which provide mechanical activity necessary for the following purposes:—the preparation of the food for digestion by reducing it to a condition of fine subdivision by means of the movements of mastication; the intimate mixing of the food with the digestive juices, so as to allow of these coming in contact with every particle; the propulsion of the food from one cavity of the canal to the next as soon as the processes of digestion in the first cavity have been completed; and finally the rejection and expulsion from the body of the indigestible portions of the food-stuffs, mixed with the products of excretion of the alimentary canal itself.

Bearing in mind the uniform character of the primitive digestive tube, one would naturally expect to find similarity in structure of the walls of the matured gastro-intestinal tube. As a matter of fact, aside from variations brought about from modifications of structure and specializations of function, this is the case. The serosa, originally almost a complete tunic, becomes very incomplete in those parts of the tube where little motion occurs, as in the duodenum, ascending and descending colon. The mucosa, primarily of uniform thickness, becomes well-developed where glandular activity is greatest, as is illustrated by the thickness of the gastric, duodenal, and jejunal mucosæ, and the relative thinness of that in the remainder of the tube. In like manner, where much work is required, the muscular tunic is well developed, and, in certain locations, is specially thickened to form sphincters. Otherwise it is thin or incomplete. In this respect, it may be compared to the musculature of the cardio-vascular system. In both of these systems the circular coat is developed to a greater extent than the longitudinal. Unusual muscular effort is required of the heart and of the stomach, and in both there is an additional layer of obliquely disposed muscular fibres. Each is the seat of rhythmical contractions. In the aorta and large arterial trunks, on the one hand, and in the duodenum and jejunum, on the other, the muscular tunics are well-developed. In the smaller arteries and the ileum the muscular tissue gradually diminishes in amount. Muscular activity in the veins and large intestine is reduced almost to a minimum, and in both the musculature is either poorly or imperfectly developed. To complete the simile, it may be mentioned that the heart and stomach are supplied both by the pneumogastric and by the sympathetic nerves, whilst the blood-vessels and intestines receive their innervation from the sympathetic system. Finally, both the heart and the stomach possess intramural ganglion-cells, which are capable of producing

spontaneous contractions of these organs, even when they are liberated from all extrinsic nervous stimuli.

Deglutition.—Mastication reduces solid food to a fine pulp, which the tongue forms into a bolus. A peristaltic contraction, which consists of contraction behind the mass with inhibition and relaxation in front of it, carries the bolus down to the stomach, which it enters through the cardiac orifice. The arrival of each bolus in the stomach can be detected by auscultating the back of a patient over the region of the cardiac orifice, which corresponds to the level of the eleventh dorsal vertebra. A gurgling sound is heard each time the food passes into the stomach. Normally this occurs from three to seven seconds after the act of swallowing (p. 56).

Movements of the Stomach.—The inhibition, which precedes the bolus, spreads to the entire gastric wall, so that the latter is now in

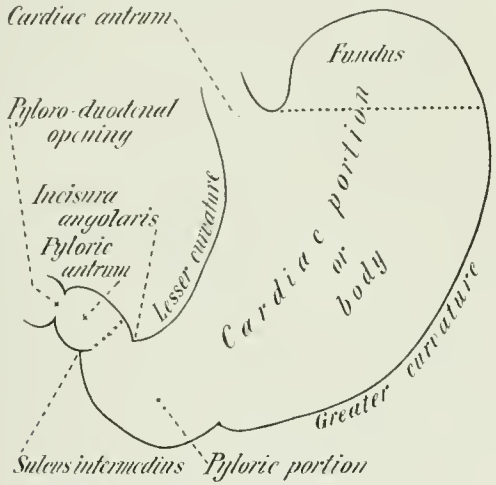

FIG. 22.—Outlines of Normal Stomach, Resting.

a passive condition for reception of the food. The food accumulates first in the fundus (Fig. 22). This arrangement is readily comprehended since, as was shown above, there is normally no empty space within the empty stomach, its cavity being only as large as its contents.

Although "appetite-juice" is present already in the stomach, yet the latter remains in a passive condition, movements appearing only after the expiration of about 30 minutes from the beginning of the meal. During this time, and probably longer, salivary digestion continues undisturbed.

The food ingested remains in the stomach for several hours, and is ejected at intervals into the duodenum. Between these intervals,

the food is isolated in the stomach from the rest of the alimentary tract by the tonic closure of the sphincters at the cardia and the pylorus.[1] The portion first ingested, lying in the cardiac portion, is marked off from the antrum by a strong constriction of the sphincter antri pylori. The fundus serves as a reservoir for the food, and is subject only to weak muscular contractions. The pylorus, on the contrary, is the seat of powerful peristaltic movements by means of which the food, received from the passive fundic reservoir, undergoes thorough churning (Fig. 23).

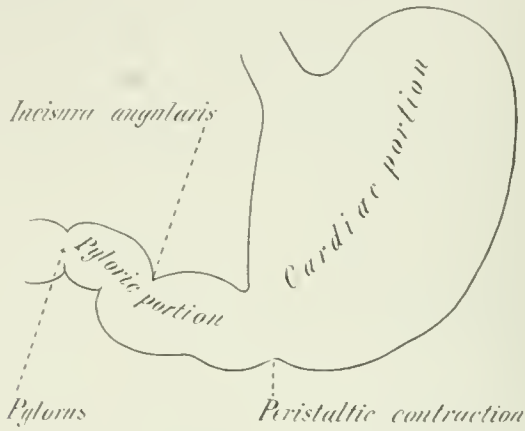

FIG. 23.—Outlines of Normal Stomach Showing Peristaltic Contraction.

After the lapse of about 30 minutes, at regular intervals, small peristaltic waves begin somewhat to the cardiac side of the sphincter antri pylori, and push the food, detached from the surface of the mass in the fundus, into the antrum pylori, made a blind pouch by the closure of the pylorus. The antrum pylori now becomes lengthened, and the peristaltic waves here increase in force as digestion progresses.

[1] Fluids ingested, however, even when the stomach already contains semisolid food, are rapidly delivered into the duodenum. It is known that in ruminants the regurgitated and chewed food is, when again swallowed, delivered directly to the duodenum by means of a channel which is temporarily formed along the lesser curvature of the stomach by contraction of the gastric musculature; thus this thoroughly masticated food is kept separate from that which still rests in the saccular stomachs awaiting its turn for regurgitation and rumination. In the human fetus the remains of such a canal along the lesser curvature may be recognized, the stomach developing as a pouched dilatation from the primitive foregut; and even in adults the gastric musculature is so arranged as to provide for the temporary segregation of a tube along the lesser curvature, which serves for the direct transmission of fluids past the gastric contents into the duodenum. This canal is named by Jefferson (1915) the *Canalis gastricus*. Roentgenological investigations confirm its existence. Moreover, only by some such mechanism as this is it possible to explain the phenomenon of *selective vomiting*, that is the vomiting of some fluid such as *bile only*, when the stomach is full of food.

The pylorus remaining closed, the food cannot escape, and is squeezed back through its sole outlet, namely, the opening in the advancing peristaltic rings, thus forming an axial reflux stream toward the cardiac end. This cycle is repeated again and again, until the hard particles of food are broken up, brought into close contact with the whole of the pyloric mucous membrane, and thoroughly mixed with the gastric juice.

At varying periods the pyloric orifice relaxes, and a few cubic centimetres of chyme are squirted, with considerable force, into the duodenum. These periods vary according to the character of the ingesta, the carbohydrates leaving the stomach first, the fats next, and the proteids last. The relaxation of the pylorus becomes more frequent as digestion progresses. When gastric digestion is over, the pylorus may open to permit the passage of undigested food-particles.

The duodenal sphincter described by Ochsner may aid in mixing the chyme just received from the stomach with the duodenal secretions and prevent its too early escape into the small intestine.

Opening of the pylorus is brought about by the presence of free acid in the stomach. In this way, the acid chyme is discharged into the the duodenum. The presence of acid in the duodenum, on the contrary, causes contraction of the pyloric sphincter, and also stimulates the flow of the alkaline pancreatic secretion. Neutralization of the acid in the duodenum gradually weakens this stimulus to pyloric closure, so that the cycle is repeated as often as is necessary for the emptying of the stomach. In this manner the intestine is charged with food very gradually by the stomach, and the gastric secretion, which inhibits the action of intestinal enzymes, is neutralized in small instalments.

The well-known experiments of Kelling (1900) which showed that filling of the duodenum inhibited contractions of the stomach, will be referred to again in connection with gastro-jejunostomy and the vicious circle.

It is not very accurately known how long food should remain in the stomach after ingestion, nor how soon the stomach should be found empty. Several hours at least must elapse; but the motor power of the stomach is said to be *delayed* if evidences of a barium (or other opaque) meal are found in the stomach on roentgenological examination 6 hours after its ingestion. The same is true if salol ingested at a certain hour cannot be detected in the urine as salicyluric acid within from forty to sixty (at most seventy-five) minutes (Ewald). Salol is not absorbed from the stomach, but by means of an alkaline

reaction in the duodenum. When a drop of the tincture of ferric chloride is added to filter paper moistened with urine containing salicyluric acid a dark brownish red or violet color is produced.

A few words may be devoted in this place to the mechanism of **vomiting,** a symptom which is of such common occurrence in diseases of the upper abdomen as to warrant the surgeon's particular attention. Vomiting is produced largely by contraction of the abdominal muscles, acting upon a fixed diaphragm, the stomach being compressed between the two. No doubt the stomach itself contracts spasmodically and aids in the ejection of the food; but this action is of comparatively trivial importance. Sensory impulses to the gastric mucosa are the chief cause of vomiting, although, as is well known, certain sights, certain tastes, and irritation of the uvula or the pharynx will in many persons have the same effect, or at least produce nausea. Sudden blockage of the urinary bladder or of the gall-bladder by a calculus may produce the same result, as may appendicular colic.

Movements of the Small Intestine.—The small intestine presents two kinds of movements, the rhythmical or pendular, which predominate, and the peristaltic.

In the pendular movements, the coils of gut sway from side to side, and, by contractions of the circular musculature, split the column of food into a number of small segments. Soon each of these segments is divided in half, and the corresponding halves of adjacent segments re-unite. This process is repeated again and again at the rate of 20 times a minute. Mall likens these contractions to those of a heart for the portal circulation. Once or twice a minute the segments are carried onward a certain distance by an advancing peristaltic wave, and collected into a new mass. Rhythmical segmentation again occurs in this new situation. In this way the food is thoroughly mixed with the digestive secretions, and every particle is brought into intimate contact with the absorptive walls, since with each constriction the mucous membrane is plunged directly into the midst of the small segments.

The peristaltic movements carry the unabsorbed material onward through the ileo-colic sphincter into the colon. Regurgitation from the colon into the ileum is prevented by the tonic contraction of the sphincter, and by the obliquity of the ileo-cecal valve.

Movements of the Large Intestine.—The presence of the semi-fluid contents in the colon starts up anti-peristaltic waves; these begin near the junction of the ascending and the transverse colon, at which point there is a physiological muscular contracture (corresponding to the ceco-colic sphincter of the lower animals), and travel slowly toward

the cecum, carrying the food into the latter. Regurgitation being impossible, part of the food must slip back in the axis of the tube, with the same effect as occurs in the pylorus. Sacculation of the large intestine heightens the efficiency of these movements. By these churning movements, the contents of the gut are mixed thoroughly with the enzymes of the small intestine, and well exposed to the actively absorbing wall of the large intestine.

Occasionally a true peristaltic wave, excited by distention of the cecum, and initiated in the latter organ, carries the food to the transverse colon. This wave, however, soon dies away, and most of the food is carried cecalward again by the anti-peristalsis. As the ascending and transverse colons are gradually filled with food from the ileum, and as absorption proceeds, the drier portions accumulate toward the splenic flexure, where they are probably separated from the more fluid parts by transverse waves of constriction, and eventually collect in the omega loop and rectum as feces.

As illustrative of the interrelation which unites the digestive processes in an orderly series of successively dependent events, Cannon sums up as follows:

"Chewing food that is relished starts the flow of gastric juice; gastric juice in the duodenum is the cause of flow of bile and the pancreatic secretion; the pancreatic secretion in turn stimulates the formation of kinase, which activates the trypsinogen. Similarly, on the motor side of digestive activities, the presence of material in the seemach normally starts gastric peristalsis; acid in the stomach seems to be the signal for the opening of the pylorus, and food is discharged; the acid food in the duodenum closes the pylorus and originates segmenting movements to churn together the food, pancreatic juice, and bile. Peristalsis carries the masses forward; now in new situations their presence occasions segmentation. Finally, the remnant of the food is forced from the ileum into the colon; and each new accession to the accumulation there, is followed by a series of antiperistaltic waves which serve to abstract still further the valuable constituents of the food. When a certain amount of useless waste has gathered in the transverse colon, forward peristaltic waves move it slowly to the rectum to be discharged."

Applied Physiology.—Above, we have given a brief account of the secretory, absorptive, and motor activities of the gastro-intestinal tract. Let us see what practical application can be made of these facts. In other woods, let us consider briefly the subject of applied physiology.

In the first instance, attention has already been directed to the

importance of a good set of *teeth*. Here, at the very entry to the gastro-intestinal tract, is a necessary set of organs, the integrity of which is essential to secure the best results from digestion. Yet too often the teeth are found incomplete in number, or the seat of varying degrees of decay. There is small wonder for this, not only because in the human race the teeth are undergoing inherited retrograde processes due to unnatural preparation of foods, but also because the dental organs are subjected to the action of the oral micro-organisms and their products, as lactic acid. Hence, we believe the teeth should be put in, and kept in, the best condition possible.

The importance of sterilizing the mouth as far as possible, and the food that enters it in the preliminary preparations for operations on the stomach, is known to all clinicians. So, too, is the regard for the teeth in the administration of hydrochloric acid.

The *stomach* is worthy of more attentive care than it usually gets. It is not just to dispose of it, from a physiological standpoint, lightly as a mere convenience for the storage of food. The bladder and the rectum, if one wish, may be considered as convenient reservoirs, but it must be remembered that these organs are the terminals for, respectively, the urinary and the digestive apparatus, and they have no other function than that of storage. The stomach, on the contrary, is at the very portals of the digestive tube. Not only does it possess definite enzymotic and motor functions, but it is also a sensitive organ, endowed with selective powers, in that it retains wholesome food, and rejects that which is detrimental or injurious. That the stomach is one of the most sensitive organs in the body cannot be denied, and it is equally true that for this reason, if for no other, it should receive much consideration, like other sensitive things. Situated between the harmful objects, bacterial, chemical, and physical, of the outside world and the delicate intestines, it exercises a distinct protective function over the gut. But it is a mistake to consider the stomach, or any other part of the gastro-intestinal tract, as a separate and distinct entity. Its embryological and physiological relations forbid this, and this is equally true of the other bodily systems, such as the nervous, the respiratory, the circulatory, and the urinary. The intimate functional interrelations of the various portions of the gastro-intestinal tract have been noted, and these inter-relations operate not only physiologically, but pathologically. Were it of any avail, we should further decry abuse of the stomach. We demand, however, that it receive consideration solely in conjunction with the remainder of the system with which it is correlated.

The great value the *appetite* holds in the interests of the economy cannot be overestimated. Like other instincts, it cannot be neglected with impunity. To restore appetite to a sickly patient is to confer upon him a boon, the effects of which are far-reaching. Not the least of these is the preservation of the integrity of the body by enriching the blood, thereby increasing the protective and defensive powers of the organism.

In the first instance, regular hours for the intake of food are as important as for the ejection of the residue from the body. Food should not be gobbled, but should be well masticated, and eaten with discrimination and care. To secure the best results, the attention should, as far as possible, be concentrated upon the process of ingestion. The degree of enjoyment with which food is taken is enhanced by the attractiveness with which it is prepared. Furthermore, the patient's tastes should receive due consideration.

Even should these conditions be fulfilled, appetite, and consequently, appetite-juice, may still be absent. In this instance, feeding would be forced; such a method is unnatural, and frequently the stomach rebels against it. Here the aim should be to restore the appetite, for this means copious secretion of gastric juice to act upon the ingesta. It often is wise to administer a cup of beef-broth shortly before meal-time, for both the water and the beef-extract contained therein are undoubtedly strong excitants of gastric juice. In the same way, suitable doses of hydrochloric acid may be carefully given, since acids are specific stimuli to the pancreas. Further, it is often beneficial to order food in small quantities, frequently repeated. By this method, too much work is not thrust suddenly upon a stomach impaired in its activity, and appetite-juice, which is so powerful, is repeatedly called forth.

For patients in whom the sense of taste is impaired, bitters may be found very useful. Not only do these stimulate the gustatory cells by means of contrast to pleasant sensations, but also they produce a certain psychic effect, and this, in turn, indirectly excites a physiological secretory activity.

Milk is a rational food for the sick, in that it nourishes the organism with the least degree of work on the part of the digestive tract, since it provokes the weakest gastric juice and the smallest amount of pancreatic fluid, and hence gives these organs rest. This rest is very desirable when the gastric glands manifest excessive activity. Alkalies, also, exert an inhibitory influence upon the digestive glands. The value of large doses of sodium bicarbonate in hyperchlorhydria is well

known. It reduces the secretion in the stomach, and, by diminishing the activity in this organ, restrains the activity of the pancreas. On the other hand, when administered in small doses, this salt, theoretically from a chemical standpoint, calls forth an increased output of hydrochloric acid.

Just as any irritating substance, bacterial, physical, or chemical evokes hypersecretion from any mucous or serous surface, so does it from the very extensive mucous membrane of the gastro-intestinal tract. This overproduction of mucus is protective in nature, in that it comes from the surface epithelium, and thus wards off the danger that threatens the more important elements of the mucous membrane beneath. Therefore, many forms of diarrhea are expressive of defence on the part of the organism.

In the same way, defensive activities in the peritoneal cavity are carried out by the omentum upon the serous tunic of the intestines. Thus, the omentum is a most important agent in developing phagocytosis and opsonins; its germinating endothelium is constantly producing lymphocytes and is capable, under proper stimulation, of throwing both newly formed phagocytes and those called from a distance into germicidal action. This wiping process is greatly aided by the vermicular and swaying movements of the intestines, which, in spite of gravity, bring all parts of the wall of the small gut in contact with the omentum, the epiploic tags having the same function for the more fixed large intestine.

CHAPTER III

SURGICAL DISEASES OF THE STOMACH AND DUODENUM

General Diagnostic Considerations

Although the diagnosis of each surgical affection is discussed in the special chapter devoted to the subject, yet it is convenient in this place to dwell upon certain general considerations. And we do this with the greater confidence, because we fear that some surgeons who are called in merely as consultants may be tempted to take the diagnosis of the malady ready made from the physician and to regard the operative treatment of their patients as the only province particularly belonging to surgery. This attitude of mind, we venture to suggest, is not only derogatory to the profession of surgery, but inimical to the ultimate interests both of patient and surgeon. While we realize that all progressive physicians are now anxious to have a surgical consultation in the case of most of the diseases of the upper abdomen which do not readily yield to hygienic and dietetic measures, we are also well aware that they are loath to consult a surgeon who can offer no other advice than to adopt the plan which is colloquially referred to as "taking off the lid." It is admitted by all that it is desirable for the surgeon to have a thorough appreciation of the natural course of the morbid process going on within the patient, in other words, that he must be well grounded in the pathology of these diseases; but it will be impossible for him to give an intelligent opinion on such questions, unless he is first abe to determine what the disease really is. To put it in the plainest terms, a consultant must be able to render an opinion which will be worth the asking; and unless his diagnostic acumen is of the highest, physicians will soon perceive that it is for their patients' interest to go elsewhere.

Anamnesis.—In every case, there is no surer foundation on which to lay the facts which go to make up a correct diagnosis, than an accurate and complete history of the patient's past medical life and present complaint. It is exceptional indeed for such a history not to point the way toward further investigations which will reveal the true malady. It may seem tedious and commonplace both to patient and surgeon to lay stress on data such as occupation, general habits

of diet, previous illnesses, etc.; but neglect of one minor detail may distort the clinical picture, and lead the diagnostician very seriously astray.

Cross-examination, and even a re-direct examination is in many cases essential to establish the truth of the patient's story.

In addition to the patient's history, the careful diagnostician will take advantage of every fact that may be learned from a complete physical examination, including *Inspection*, *Palpation*, *Percussion*, and *Auscultation; Mensuration* also may be of value in a few instances. Nor should laboratory aids be despised (p. 57). *Skiagraphy* as an aid to diagnosis is discussed at page 61.

Inspection.—The patient should lie flat on the back, with the entire abdomen and lower thorax bared, and in a good light. The general contour of the abdomen should be noted, whether fat or emaciated, tense or flaccid; together with the presence or absence of localized bulging, of peristaltic waves, of scars from previous operations, etc. Attention should be directed to the character of the breathing, whether it be natural or affected by the surgeon's examination. The appearance of the costal angle should be noted, as well as any undue bulging or depression of the pit of the stomach. The configuration of the thorax, as indicative of tight lacing, may prove of diagnostic value in certain affections of the liver and stomach.

Palpation.—This is the most valuable of all means of physical examination at the command of the surgeon. It should never be omitted, and should always follow inspection. In order to obtain the greatest number of diagnostic points by its means, it is important to have the abdominal muscles as relaxed as possible. This relaxation is best secured by having the patient's head and shoulders slightly elevated, and by flexing the thighs on the abdomen. The thighs should be passively, not actively, flexed; so that it is best either to support the knees by placing a pillow beneath them, or to have the thighs flexed so far that the soles of the feet will rest comfortably on the bed or couch on which the patient lies.

The hands of the diagnostician should be warm, so that no reflex spasm of the abdominal muscles will be caused by chilling from contact of the hands. It should be an invariable rule to begin the palpation in some presumably healthy region of the abdomen, in order to accustom the patient to the palpating hand before the diseased area is reached, and to ascertain, if possible, the natural condition of the belly wall in health. The patient's attention may be diverted from the local examination by conversation. The entire abdomen should be

thoroughly examined by palpation, the hand being allowed to slide over its surface rather than being raised and again laid on the skin, for fear of producing involuntary contraction of the abdominal muscles from the new contact.

The degree of rigidity in all portions of the abdomen must be noted. Attentive examination should be made of any rumor detected, noting especially its location, its consistence, its general conformation, its mobility, and if any, in what direction; the presence or absence of movement during respiration; and finally its relation to surrounding structures must be considered.

The degree of tenderness present in the various portions of the abdomen must be noted, together with the amount of pressure necessary to provoke it. The degree of tenderness to pressure, combined with the rigidity of the abdominal wall at the site of the tenderness, will often be of marked diagnostic significance.

The presence or absence of a succussion splash in the stomach may be determined by means of palpation. This splash is very significant in cases of gastrectasis if found in certain relation with meals, as it will indicate decided lack of gastric motility.

Percussion.—This is of the utmost value in mapping out the relation of the various abdominal organs. It will reveal the size and position of the liver, the position of the colon, of the stomach, and of the spleen; the presence or absence of hepatic dullness; the presence or absence of free fluid in the abdominal cavity, etc. In many instances much additional information is to be gained by distending the stomach with air or liquid, so that its position and extent can be definitely determined. The stomach may readily be filled with water by drinking; this method is so extremely simple that it often is overlooked. Air may be forced into the stomach by means of a hand bulb on the stomach tube; we do not approve of the use of an effervescing powder for inflation of the stomach, because this latter method cannot be controlled, and may lead to serious results; moreover, it certainly causes much more discomfort to the patient than does the use of a stomach tube, by means of which the amount of air introduced can be accurately regulated. The patient himself is the best judge of the amount of air to be introduced; and the least discomfort on his part should make the surgeon desist from introducing more air. The same is true for the introduction of fluids, either for the purpose of distending the stomach, or for washing it out. If any doubt as to the outlines of the stomach remains after distention with air or fluid has been tried, it is at times advisable to inflate the colon with air while the stomach is distended

with fluid. In this way percussion will reveal their relative positions with great accuracy, and may throw much light on an obscure condition, or reveal the exact location of any tumor that may be present.

Auscultation.—Auscultation alone, or in combination with percussion, may be of decided value in the diagnosis of diseases of the upper abdomen. It is well known that the time when food or liquids enter the stomach can be detected by auscultating the thorax about three inches below the angle of the left scapula. It is well for the inexperienced to listen to the normal sound in healthy individuals, many times, before attemping to diagnosticate a lesion of the esophagus or cardia by this method. The amphoric, rushing sound is heard from three to seven seconds after the act of swallowing; if delayed more than ten seconds, it is safe to infer that some obstruction, spasmodic or organic, is present.

Auscultatory percussion is sometimes of aid in determining the exact outlines of a distended stomach. In a similar manner, the coin test may be used in the case of an air-containing cavity, such as the stomach, or in that of a subphrenic abscess in direct connection with the gastro-intestinal tract.

Mensuration.—This is occasionally useful to record from time to time variations in the amount of abdominal distention; to note increase of ascitic fluid; to compare one side of the abdomen with the other, etc. Hoover's test for subphrenic abscess is described at p. 408.

General Health.—In deciding for or against an operation it is of course requisite for the surgeon to take into consideration the general health of the patient. The state of the heart, the lungs, and the vascular system should be attentively studied; and careful examination should be made of the amount and quality of the urine excreted. Myocardial disease will be in general more of a contraindication to operation than will a well compensated valvular lesion. High blood pressure is by no means a contraindication to an otherwise necessary operation; but this factor, as well as the renal functions as determined by the phenolsulphonephthalein test are of great value in prognosis, and of much importance in the choice of an anesthetic. The gastric and intestinal manifestations of nephritis should be kept constantly in mind.

Gastric Analysis.—Chemical analysis of the gastric secretion, and determination of the motor functions of the organ, sometimes are of aid in reaching a correct diagnosis.

The *motor function* of the stomach is best tested by means of the fluoroscope (p. 64). If this is impossible, the stomach may be evacu-

ated by the stomach tube about twelve hours after the ingestion of an ordinary full meal. It is better, as advised by Paterson, to give a definite quantity (12 oz., or 340 cc.) of milk on an empty stomach and to evacuate the stomach exactly ten hours later. It is convenient to give some raisins with the milk, as the skins are readily recognized if recovered. If food-remains are recovered it indicates pyloric stenosis or motor insufficiency. Any amount even up to 400 cc. may be recovered. If no food-remains are found, there is no pyloric stenosis. If acid fluid without food-remains is recovered, this is an indication of hypersecretion, which occurs in duodenal ulcer, without pyloric stenosis, as well as in many cases of chronic appendicitis, and in a few cases of gastric ulcer.

For purposes of *chemical analysis* a uniform test meal should be employed. The usual test meal (Ewald's) consists of one or two slices (60 grammes) of toast and a cup (250–400 cc.) of weak tea, with sugar but without milk. Whatever the nature of the meal, it cannot too strongly be emphasized that it should be given on an empty stomach (as for instance just after lavage to test the gastric motor functions) and that it should be constant in amount and quality if the results of the analysis are to be compared with those obtained in other cases. The stomach contents are drawn off exactly one hour after the patient began to eat his test meal. The amount normally recovered is from 100 to 140 cc. If there is stenosis this amount may reach even to 500 or 600 cc. If the case is one of duodenal ulcer, often less than the normal amount is recovered.

It is not worth while in a work of this kind to go into any detailed statement of the chemical tests in common use in gastric analysis. A summary of the usual findings, however, may be of use. These we have condensed from Paterson's valuable monograph on the Surgery of the Stomach.

Free hydrochloric acid is *present* in duodenal or pyloric ulcer, in gall-bladder diseases, and *sometimes* in chronic appendicitis. It is *absent* in malignancy, in severe chronic gastritis, in hour-glass stomach, and *usually* in chronic appendicitis.

Lactic acid is almost always present in small amount after the ordinary test meal. Only if present after a special (Boas) test meal, is its presence significant: At night wash the stomach absolutely clear. Next morning wash it again, and then give oatmeal soup (one tablespoonful of rolled oats in 1000 cc. of water, boiled down to 500 cc., to which is added a little milk but *no salt*). Two hours later draw off the stomach contents, filter, and test for lactic acid with Uffelmann's reagent. Under such circumstances *lactic acid is rarely present*

except in gastric carcinoma. A positive test therefore is of great significance.

The *total acidity* (HCl, volatile acids, and acid phosphates) of the gastric juice varies in health between 55 and 65;[1] a figure over 70 is regarded as hyperacidity, and one under 50 as hypoacidity. Hyperacidity should not be confused with hyperchlorhydria: as the total acidity is contributed to by HCl, by volatile acids, and by acid phosphates, hyperacidity may be due to an increase of any or all of these constituents. Hyperchlorhydria implies an increase solely in the hydrochloric acid.

The amount of free HCl normally varies from 0.018 to 0.022, the average being 0.020.[2]

In duodenal ulcer it is *increased.*

In gastric (pyloric) ulcer it is *increased.*

In pyloric stenosis it is *decreased.*

In gastric ulcer which has cicatrized or which is in the body of the stomach, it is *decreased.*

In hour glass stomach it is *decreased.*

In carcinoma it is *absent.*

In chronic appendicitis it is *decreased* or *absent.*

The amount of total chlorides normally varies from 0.310 to 0.330, the average being 0.320.

In duodenal ulcer there is a *very marked increase.*

In gastric ulcer there is usually a moderate *increase,* especially if the ulcer is near the pylorus.

In carcinoma it is *decreased.*

In pyloric stenosis it is usually *decreased.*

In chronic appendicitis it may be increased or decreased, usually decreased.

The amount of the volatile acids normally varies from 2 to 5.

In gastric ulcer there is no change.

In duodenal ulcer usually there is no change.

In carcinoma usually an increase.

In chronic appendicitis usually an increase.

[1] That is, it requires from 55 to 65 cc. of decinormal sodium hydroxide solution to neutralize the acidity in 100 cc. of gastric contents.

[2] Recent investigations by Boldyreff (1914) tend to demonstrate that the real acidity of fresh gastric juice is equivalent to 0.5% HCl, much the same as in dogs; but as usually obtained in man it has been neutralized in part automatically by reflux of alkaline duodenal secretions, and in part by the test meal administered. This raises the question whether "clinical hyperacidity" may not be due to loss of duodenal reflux rather than to a pathological state of the gastric juice.

The amount of the protein hydrochloric acid normally varies from 0.220 to 0.250.

In carcinoma it is *very markedly diminished*, usually less than 0.100.

In other conditions it is of no significance.

The amount of the mineral chlorides is *very markedly increased* in carcinoma (the free HCl probably is fixed by the alkaline secretion from the growth).

The following table, adapted from Paterson's, gives certain typical gastric analyses:

Typical Gastric Analyses

Lesion	Total acidity	Total chlorides	Free HCl	Protein HCl	Mineral chlorides
Duodenal ulcer	82	0.350	0.038	0.229	0.080
Duodenal ulcer	80	0.405	0.055	0.275	0.075
Gastric ulcer (pyloric)	75	0.325	0.040	0.205	0.080
Gastric ulcer (mid-gastric)	78	0.308	0.025	0.206	0.077
Gastric ulcer (hour-glass)	70	0.332	0.000	0.230	0.098
Carcinoma	28	0.178	0.000	0.051	0.124
Appendicular gastralgia	61	0.288	0.007	0.201	0.080
Normal Average	60	0.320	0.020	0.235	

Passing the Stomach Tube.—The stomach tube should be in good condition, and its surface should be perfectly smooth. As soon as the surface begins to crack, the tube should be discarded. The tube should be cleansed and boiled after using; it should then be dried in the air and put away. It is not necessary to boil it again just before using, though it is often desirable to do so; but repeated boiling soon destroys it. The tube should be marked about 40 or 45 cm. from its extremity, and when introduced this mark should correspond to the patient's teeth. The esophagus begins about 15 cm. from the dental margin and is about 25 cm. in length. When the tube is to be used as a pump it should have a hand bulb attached to it about 25 cm. beyond the "tooth mark." If the delivery tube is pinched in the fingers close to the bulb, pressure on the bulb will force its contained air into the stomach; this air will be held in the stomach by pinching the stomach tube as the bulb is allowed to expand. Air will escape spontaneously

from the inflated stomach, through the tube, as soon as the bulb is detached. To withdraw fluid from the stomach, syphonage will usually suffice; but by reversing the maneuvres above described, the apparatus may be converted into a suction pump (Fig. 24).

If the patient is strong enough, it is more convenient to pass the tube while he is in a sitting posture; if necessary, however, it is quite possible to introduce it while he is lying down. In most cases suffi-

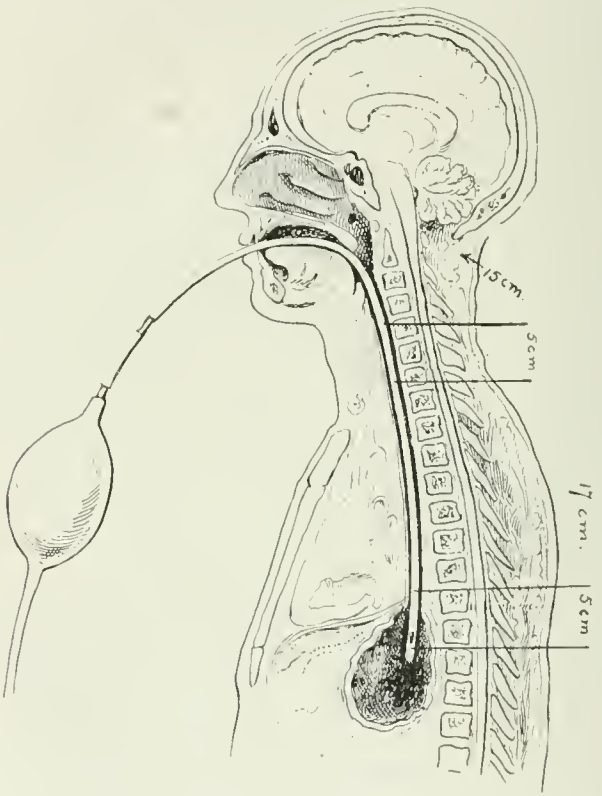

FIG. 24.—Diagram of the Stomach Tube *in situ*.

cient lubrication will be afforded by the mucus in the pharynx and esophagus, the tube being chilled by being laid in a basin filled with ice-water just before being introduced. Oils or other lubricants are usually very disagreeable to the patient. A mouth gag need not be employed except in children and unconscious or refractory patients.

The surgeon, facing the patient, and having his own clothing, as well as that of the patient, suitably protected, takes the stomach tube, about six inches from its end, in his right hand, holding it as a pen; then, directing the patient to open the mouth widely, he passes the

index finger of his left hand into the patient's mouth. With the left forefinger as a guide, and drawing forward on the base of the tongue, the stomach tube is gently, but firmly passed back until its end touches the posterior wall of the pharynx. By then guiding its point downward with the left index finger, it is gradually fed onward by the right hand; and by keeping it close against the posterior pharyngeal wall, it will glide into the esophagus. At this moment the patient, if the stomach tube is being passed for the first time, usually gags, and feels as though he were smothering. If he is assured, however, that all is going well, his momentary distress is quieted, and if he takes deep breaths and swallows frequently the tube will glide down into the stomach, until the tooth mark reaches the dental margin. It is very seldom that force is required, when once the end of the tube has become fairly engaged in the esophagus; and if the surgeon is gentle and patient in his manipulations, the patient will find that the ordeal of having a stomach tube passed is by no means unbearable; indeed that it is much less disturbing in reality than in anticipation.

After a patient has had a tube passed once or twice, no guiding finger will be necessary, and the patient very often will prefer to pass it himself, instead of having this done by the surgeon. Those who are in the habit of having stomach tubes passed experience very little more discomfort than is felt in passing a soft rubber catheter through a normal urethra.

Lavage of the Stomach.—Tepid water, either alone, or with a little bicarbonate of soda added, is the liquid that is usually employed. For patients with marked fermentative changes, a weak solution of permanganate of potassium is useful. Not more than 250 to 300 cc. should be passed into the stomach at first. This should be done very gradually, with the funnel of the tube very little higher than the level of the stomach. The feeling of beginning discomfort on the patient's part is the safest indication of the amount to be introduced at one time. In unconscious patients, and in those with marked disease of the stomach, only a very small quantity should be used at any one time; and even greater gentleness than usual should be employed, on account of the danger of producing rupture of the stomach (see p. 297). After the proper amount has been introduced into the stomach, the funnel should be lowered and then inverted over a waste bucket, and the gastric contents syphoned off, the process being repeated until the fluid returns clear.

The X-rays in Diagnosis of Lesions of the Upper Abdomen. In cases where the clinical history and ordinary methods of physical

examination leave the diagnosis, in doubt, or where it is desired to obtain confirmation of diagnosis. roentgenology often will be of material assistance. There is but one objection to the use of the X-ray in this connection, and this is the fact that it is useless unless employed by an expert in this particular branch of X-ray work. The technique employed is so complicated, and demands such close study, that a description of it would be entirely out of place here; but it is necessary for the clinician who is to take advantage of work being done by ex-

FIG. 25.—Normal Peristalsis, Resembling Indurated Ulcer. (*Lewis Gregory Cole, in American Journal of Roentgenology, November*, 1915.)

pert Roentgenologists to realize what aid may be given them, and what preliminary preparation of the patient is necessary to render the skiagraphic examination of value.

When possible, the patient's gastro-intestinal tract should be thoroughly emptied. This is best accomplished by means of a purge and abstinence from food. Liquids may be administered if necessary, but even this diet should be limited to broths, albumen water, etc. Milk is apt to form curds, and these may be very misleading. The

patient should be prepared to remove the clothing from the abdomen. It is generally better to remove all clothing, substituting a dressing gown without buttons, or a sheet which will cover the entire body.

Not only is the technique of making the radiographs of the alimentary tract difficult, but the interpretation of the findings is also a matter of expert knowledge. Study of Fig. 25, which is a normal, and of Fig. 26 a pathological stomach, will convince anyone of the ease with which an error may be made.

More information of value can probably be obtained from a

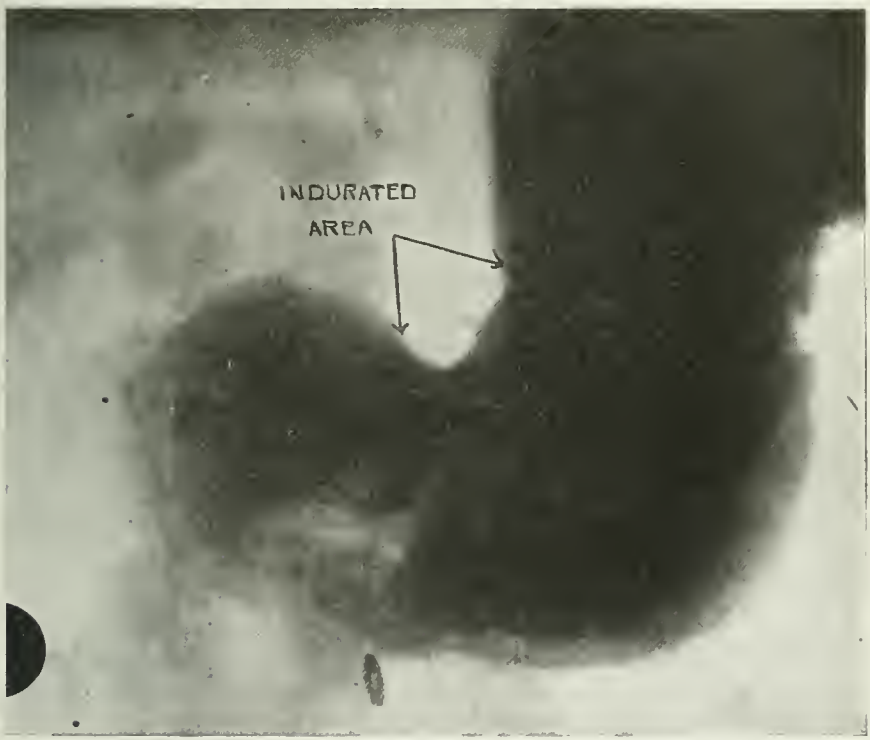

FIG. 26.—Slightly Indurated Ulcer with Slit-like Crater. From a Patient under the Care of the Senior Author. (*Lewis Gregory Cole, in American Journal of Roentgenology, November*, 1915.)

roentgenologic examination of the **stomach,** than from that of any other viscus in the upper abdomen. This organ is studied after it has been rendered transparent by inflating it with air, or after rendering it opaque by the ingestion of some bismuth or barium preparation. Air, as a rule, does not distribute itself well in the stomach, and is really important only in the study of the fundus or "upper pole."

The stomach normally, and except when adherent to surrounding

structures, is distinctly movable and changes its contour and position with the position of the patient's body. The posture of the patient, therefore, should vary according to the portion of the stomach which it is desired to examine. The standing posture generally is the most useful. In this position the axis of the stomach is almost vertical and the organ is situated almost entirely to the left of the median line. The lower one-third is more or less horizontal, but the upper two-thirds are nearly vertical in this position. The upper pole normally rests against the diaphragm; the lower pole in the standing position reaches to the umbilicus or lower and is little lower than the pylorus. In gastroptosis the lower pole of the stomach may reach any distance below this level. In the dorsal decubitus the greater portion of the stomach occupies the left hypochondrium. Most text-book illustrations are made with the viscera in position as seen on the operating or dissecting table, and hence do not give an accurate idea of the position assumed by the stomach when the patient is erect.

A portion of barium mixture may be administered with the patient standing. By fluoroscopic examination a swallow of liquid food may be seen to enter the stomach completely in about seven seconds. A delay usually means some constriction at the cardiac orifice, either spasmodic or organic. If this constriction is organic, solid portions of food, or bismuth capsules or pills, will be retained a longer time, according to their size, the calibre of the constriction, and the degree of solubility of the ingested material. If due to malignant change, the constriction usually can be outlined in the gaseous field which occupies the upper portion of the stomach when the patient is in a standing posture.

A constriction of the **duodenum** at times may be shown by a dilatation of the portion preceding the stricture. A duodenal ulcer may be detected by signs analogous to those of gastric ulcer, especially if a niche is visible.

The **solid organs** of the upper abdomen cannot be so easily examined by means of the X-ray; but Dandy (1919), Stewart, and others have shown that in cases of intestinal perforation, with escape of air into the peritoneal cavity, the outlines of the liver and diaphragm may be clearly seen in skiagraphs; and that in dogs, the experimental injection of 500 cc. of air into the peritoneum renders visible practically all the intra-abdominal organs, and appears to do the animals no harm. The method has been employed occasionally also in man.

Lesions of the stomach itself may also be detected by fluoroscopy

though some roentgenologists maintain that a series of X-ray plates, made in as rapid succession as possible (serial roentgenography), is preferable.

Delayed emptying of the stomach, evidenced by the presence of a residue from the opaque meal more than six hours after its ingestion, is one of the most valuable and readily detected signs of disease, particularly of ulcer or carcinoma; but it may indicate nothing more than retention from gastroptosis. Too rapid emptying of the stomach occurs in some cases of duodenal ulcer, and is associated with hyperperistalsis.

A gastric ulcer of comparatively recent formation may be detected by observing that there is a constantly localized obstruction to the normal peristaltic waves (Cole, 1915). An *ulcer of long standing*, which has become callous and indurated, presents (in addition to the constant interference with peristalsis at a given point) also a shadow cast by the opaque material which fills its crater; this shadow is known as Haudek's niche. If a chronic perforation (p. 84) is present the shadow of the accessory pocket may also be visible in the skiagraph. When the gastric ulcer is at or near the lesser curvature, as is usually the case, there is nearly always a constriction in the neighboring gastric wall which shows itself as an "incisura" at the greater curvature. According to Carman, a true incisura should be subjected to and withstand the following tests: (1) It must be constant and stationary; (2) it must be present when the stomach hangs normally; (3) it must survive vigorous palpation; and (4) it must persist after the patient has been given an antispasmodic to physiologic effect (as, for example, tincture of belladonna, 10 drops, three times daily for two or three days or until flushing of the skin and disturbance of vision results). A deep incisura may be mistaken for the contraction of a true *hour-glass stomach*.

A carcinomatous ulcer can with difficulty be distinguished from a benign callous ulcer,[1] but a *well developed carcinoma* presents fairly characteristic roentgenologic signs, especially a filling defect at one portion of the outline, due to the projection of the tumor into the cavity of the stomach.

[1] Other competent roentgenologists do not agree with Cole, who categorically asserts: "We can state that a patient has not a cancer of the stomach with as great a degree of certainty as we can state that he has not a fracture of the hip." (N. Y. Med. Jour., 1915, ii, 26.)

CHAPTER IV

GASTRIC ULCER

Pathogenesis.—Ulceration of the stomach may occur as the result of tuberculous, syphilitic, or malignant disease, but the form which concerns us in this chapter has no such specific cause. Though gastric ulcers have certain very marked characteristics which distinguish them from many other ulcers, there is really little that is mysterious about them. They are due to the same causes as ulcers elsewhere in the body, and seem to owe their peculiarities only to their situation.

Their pathogenesis has been studied especially by Dieulafoy, by Gandy, by Hort, and lastly by Bolton. From the Paris Thesis of Gandy (1899), which exposes the views of Dieulafoy, and from the recent excellent monograph of Bolton, much of what follows is derived.

Gandy showed that in nearly all toxemias there are gastro-intestinal ulcers, and that in almost all cases of gastro-intestinal ulceration there is present some form of toxemia. He pointed out the remarkable similarity which exists between the ulcers of toxemias (including the intestinal ulcers met with in cases of burns), and the so-called simple ulcers of the stomach. They are alike in latency, in tendency to hemorrhage and perforation, and in their acute formation. They are also alike in their clinical course: the earliest stage in all is ecchymosis; then hemorrhagic infarct; slough; hemorrhagic erosion; "ex-ulceratio simplex" of mucosa; true ulceration with hemorrhagic borders; and finally perforating ulcer, or chronic ulcer with thickened border, or a cicatrix. He was able to trace these forms in (1) burns; (2) infantile diseases (melena); (3) infections (erysipelas, septicemia, pyemia, local septic infections, variola, scarlatina, purpura, puerperal infection, infections of uterus and annexa, infections of genito-urinary apparatus, strangulated hernia, biliary infections, pneumonia, pleural infections, phthisis, diphtheria, articular rheumatism, rabies, tetanus, cholera, dysentery, typhoid fever, etc.). In infantile diseases, burns, and typhoid fever he was able to find pathological changes representing each of the degrees above mentioned, beginning with ecchymosis, and ending with perforation. He remarks, further, that observers have for many years noted all the above lesions in other parts of the stomach or digestive tube, in cases of gastric ulcer, but do not seem

to have appreciated the fact that they probably represented earlier stages of the same process. In patients with gastric ulcer it is probable that the toxemia is always of infectious origin; and previous lesions of the liver and kidneys, by increasing the toxemia, may act as predisposing causes.

Somewhat similar views have been expressed by Hort. With the advantages which the most recent researches in chemical biology have placed in the physician's power, Hort has been able to carry the theory of the toxemic origin of gastric and duodenal ulcers one step further. He thinks gastric ulcer, including, as above stated, ecchymosis, erosion and actual ulcer, is due to a general blood disease, in the nature of a toxemia, the local effects in the stomach being due to the production of hemorrhagins, which eat through the endothelial lining of the blood-vessels; and secondly to mucolysins, which destroy the gastric mucosa.

Clinical experience, that best of teachers, has been indicating during the past few years that in a very large number of cases of gastric ulcer the original focus of infection lies in the *appendix vermiformis*[1] and the investigations of Rosenow, as to the elective localization of streptococci, are a further confirmation of the view of the infectious origin of gastric ulcers and similar lesions. These studies indicate that the cells of the tissues for which a given strain of bacteria shows elective affinity may "take bacteria out of the circulation as if by a magnet—adsorption."

Bolton has shown by careful histological studies, that the *initial lesion* in gastric ulcer is (a) localized necrosis of the mucous membrane; (b) localized hemorrhage in the mucous membrane; or (c) inflammation of the lymphatic follicles at the bases of the gastric glands, upon the muscularis mucosæ. *Necrosis*, he points out, is due usually to bacteria or their toxins circulating in the blood stream, and not to the food contents of the stomach; poisons of metabolic origin, such as the gastrotoxin described by Bolton himself, act in a similar manner. The cells of the gastric mucosa being primarily injured by circulating poisons, necrosis is readily produced by the local action of the gastric juice. Necrosis may arise in this way without any preceding hemorrhagic

[1] Moynihan (1910) was one of the earliest to call attention to the relation which exists between appendicitis and gastric lesions. One of us (Ashhurst) has narrated elsewhere (1914) several interesting cases in which the relation was too close to be easily overlooked: (1) occurrence of gastric perforation just one year after removal of an acutely inflamed appendix; (2) occurrence of gastric symptoms three years after removal of a gangrenous appendix, and the finding of a subacute perforation of the stomach at the second operation. Indeed, the more often the abdomen is opened for gastric symptoms, without neglecting at the same time to investigate the condition of the appendix, the rarer does it become not to find gross evidence of disease in the latter structure.

change or lymphatic inflammation. *Preceding hemorrhage*, however, is actually a frequent cause of gastric ulcer, and is due to bacterial toxins circulating in the blood stream and destroying the endothelial cells of the capillaries, thus paving the way for the local destructive action of the gastric juice. *Inflammation of the lymphatic follicles* in the stomach has been recognized since the time of Cruveilhier (1835–42). Though solitary follicles occur all over the stomach, between the mucosa and the muscularis mucosæ, they are most thickly studded along the lesser curvature, and especially toward the pylorus. The formation in one or more, perhaps in very many, of these follicles, of a submucous abscess, followed by its rupture into the gastric cavity, allows the gastric juice to act on the base of the ulceration thus exposed.

An ulceration formed in one of the ways above indicated would soon heal in other situations, or in a normal stomach; but when constantly exposed to the action of the gastric juice (especially if hyperacid) it shows no tendency to heal, but rather to extend. It extends faster (sometimes exceedingly fast) if there is pyloric obstruction, or if there is any systemic or localized infection. In such cases sudden hemorrhage or perforation may be the first indication of gastric disease.

A few words should be said in this place in regard to what may perhaps be called the *mechanical theory* of the pathogenesis of gastric and duodenal ulcers. It is mentioned also in Chapter XVI in connection with the stagnant gall-bladder.

This theory has been ably presented in a recent paper of great interest by Waugh (1920). He points out that Lane's theories of toxic absorption as a result of intestinal stasis being the cause of varied lesions have not met with general acceptance. And Waugh suggests that a congenitally mobile cecum and ascending colon may by their drag exert injurious traction upon certain of the upper abdominal organs (stomach, pylorus, gall-bladder) if these are well fixed; whereas if they are not well fixed, gastroptosis, etc., will result. Now he proposes the theory that such points of injurious traction in the pyloric region of the stomach or the duodenum form places of lessened resistance in which the ulcers are prone to develop. This theory thus obviates the necessity for assuming a toxemia from chronic intestinal stasis, which has never been proved to exist. Waugh points out, moreover, that in these cases (*cæcum mobile*) surgical treatment should be preventive, as the ulcers and other lesions seen in adult life are end results and cannot be cured by removal of the cause. True preventive measures, he contends, consist in proper fixation of the cecum and ascend-colon in childhood.

Pathology.—There is very little doubt that every so-called chronic or callous or indurated ulcer was at one time acute, and began in one of the ways above described. By gross inspection we may recognize the two following main classes of lesions: (*a*) *Erosions;* and (*b*) *Ulcer* or *Ulcus.*

(*a*) *Erosions* ("Follicular erosions" of Cruveilhier; "punctate erosions" of Brinton).—Under this heading may be recognized recently formed (acute) lesions of the gastric mucosa, so small as often to be found only after considerable search, and appearing as mere abrasions of the mucous membrane. They extend no deeper than the umscularis mucosæ, seldom or never cause symptoms, and probably in the vast majority of cases heal promptly without leaving any scars. The "exulceratio simplex" described by Dieulafoy may be regarded as a slightly more advanced form; it includes the muscularis mucosæ. *Ulcerations* as distinguished from erosions, may involve the entire thickness of the gastric wall sometimes give rise to alarming hemorrhages. and may perforate. They may coincide with the erosions, and are probably due to the same cause. They are not palpable from the serous surface of the stomach, but heal with the formation of slight cicatrices, which often may be recognized at operation or autopsy. These scars are more frequent in the cardiac half of the stomach showing that here they heal more readily, while near the pylorus the unhealed ulcerations are more frequently found.

(*b*) *Ulcer or Ulcus* ("Simple, "round," or "peptic" ulcer).—If the ulcerations just described do not heal soon, they become indistinguishable from "ulcus" which is the form commonly intended when "gastric ulcer" is mentioned. The recent formation of such an ulcer is frequently indicated by the presence of a partly adherent black slough (Plate I, a). the black color being due to the action of the gastric hydrochloric acid on the hemoglobin in the ulcer. The most recent ulcers of this type are the classical round, open "punched out" gastric ulcers. If healing is delayed, the edges of the ulcer become sloping, the ulcer grows more or less conical in shape, its borders are thickened, and the surrounding mucosa becomes puckered up (Plate I, b). Bolton's studies further show that as long as the ulcer is extending in area its edges are undermined and overhanging, that the submucosa is thickened, and that the sloughs, formed at the expense of the submucous coat, show evidence of digestion at their edges by the gastric juice. The final stage, in which healing is probably impossible, is that of chronic, callous, indurated ulcer, with thick, hard, raised, undermined borders, and with its base covered with tenacious mucus.

(a) Chronic Gastric Ulcer on Lesser Curvature; Specimen Secured by Excision. Note the Thickened, Edematous Gastric Wall, the Enlarged Lymphnodes in the Attached Omentum, and the Recent Clot Covering the Ulcer's Base. Female, 39 Years, in Good Health 2 Years Later. Path. No. 8888. *Lankenau Hospital.*

(b) Callous Gastric Ulcer on Posterior Wall near Lesser Curvature; Specimen Secured by Excision. Note the Radiating Cicatricial Bands, the Sclerosed Margins of the Ulcer, Its Base Covered with Adherent Slough, and the Thickened Edematous Gastric Wall. Male, 63 Years, in Good Health 2 Years Later. Path. No. 8774. *Lankenau Hospital.*

Face p. 70

Among 143 specimens of ulcer (48 gastric and 95 duodenal) excised by the senior author and studied microscopically by Reiman (1920), 22 were classed as acute (5 gastric and 17 duodenal), 91 as chronic (29 gastric and 62 duodenal); while 30 ulcers (14 gastric and 16 duodenal) were chronic but gave evidence of recent acute exacerbations.

Clinical Pathology.—Although *erosions* and *ulcerations* almost invariably are multiple, the true gastric *ulcer* is single in about 77 per cent. of cases, and the chronic callous ulcer is single in from 80 to 90 per cent. of cases (Bolton). In more than half the cases there are multiple small white scars, left by healed ulcerations. The *site* of the ulcer usually is near the lesser curvature, and much more often in

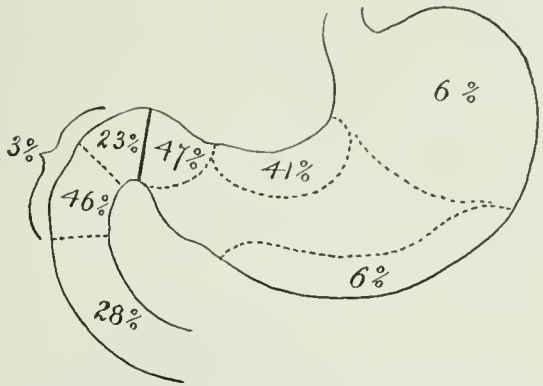

Fig. 27.—Diagram Indicating in Percentage, the Sites of 268 Ulcers (53 Gastric and 215 Duodenal), Recently Encountered at Operation by the Senior Author. In 3 per cent. of the Cases of Duodenal Ulcer, Two Ulcers Were Found, One in the First, the Other in the Second Part of the Duodenum. (*Lankenau Hospital.*)

the pyloric region of the stomach than elsewhere. Probably this is owing to the large number of lymph follicles along the lesser curvature, and because ulcerations in other parts of the stomach show little tendency to remain unhealed. Scars of healed ulcerations are much more often encountered in the body and cardiac portions of the stomach than in the pyloric region or along the lesser curvature. Unhealed ulcers are also more frequent on the posterior than on the anterior wall of the stomach. The existence of similar ulcers on the anterior and posterior walls near the lesser curvature (the so-called "kissing ulcer") is more probably due to similar relations to the blood supply than to any fancied infection of another portion of the stomach by an existing ulcer. Figure 27 indicates the sites of 268 ulcers (53 gastric and 215 duodenal) recently encountered at operation by the senior author.

The *course* pursued by gastric ulcers does not differ materially from that of ulcers of any other region exposed to such constant injury. If the early symptoms are sufficiently severe to compel attention and procure for the patient appropriate medical treatment, the ulcers usually will heal, it is believed, leaving only insignificant cicatrices. If, however, the symptoms at the onset are not very severe, proper treatment is neglected, and the lesions, especially those along the lesser curvature and near the pylorus, develop into true round "open" ulcers. Ulcers of the stomach have been compared to leg ulcers and the resemblance is in many respects close. Both develop insidiously, and are endured by the patient because not very acute in character, and because proper treatment would necessitate interruption of the usual activities of life. The longer gastric ulcers remain unhealed, the longer time will be required for their repair by medical means; indeed, it is doubtful whether a true gastric ulcer, as distinguished from an erosion, is ever healed under purely medical treatment.

FIG. 28.—Ulcer on the Lesser Curvature; a Very Frequent Site.

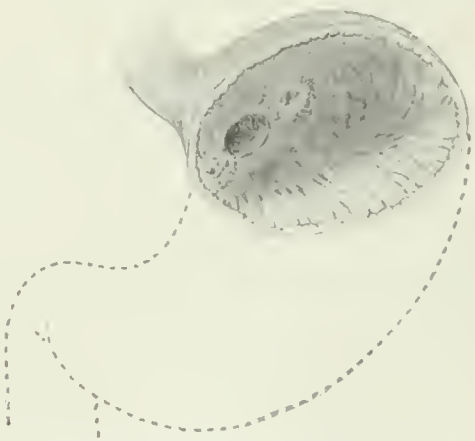

FIG. 29.—Twin Ulcers Each Side of the Cardiac Orifice. A Very Unusual Site. From a Patient in the Lankenau Hospital.

Though latency may be procured by these means, recurrence after medical "cure" is frequent; and it must be frankly confessed that even surgical operations are not sure preventatives of recurrence, since they at most place the patient in the same condition with respect

to future gastric ulcers as he was before the development of the ulcer for which the operation was done.

When a simple ulcer has once formed in the stomach it shows no innate tendency to heal. Its tendency, on the contrary, is to remain unhealed, and eventually to become callous. At any time after its first appearance as an erosion it may give rise to the acute complications, hemorrhage or perforation. As far as duration alone is concerned, it soon becomes a "chronic" ulcer whatever its special characteristics may be. But it is not always possible merely by looking at an ulcer to determine its duration; and when several ulcers co-exist, it is often manifest that some of them are much more recent than others. Nor does the duration of the clinical symptoms always decide upon the age of the ulcer. It is frequently impossible to tell which one of several ulcers has caused symptoms the longest. An ulcer may have been latent for a considerable period before producing symptoms, or may have become quiescent some time since, and the recent symptoms may have been caused by a more recently developed ulcer, or by one which, though present for a long time, only recently has become symptom producing. As a rule, however, it is safe to assume that an ulcer with callous edges, and with its margin shelving rather than punched out, is an ulcer of long duration—possibly ten to fifteen years; while an ulcer resembling in character the "exulceratio simplex" of Dieulafoy is manifestly of quite recent formation. The "acute round ulcer," simple or peptic ulcer, which appears cut out of the stomach wall, is of indefinite duration, and while we can say that it has not existed as long as an ulcer with callous margins, we cannot be certain that it has existed longer than a few months or even weeks.

This simple "punched out" ulcer is still actively ulcerating, and has not commenced to granulate to any appreciable degree. Its base is formed by the muscularis of the gastric wall, and it is much more prone to perforation than is an ulcer of the callous type. And when perforation takes place in this punched out ulcer, the peritoneal cavity is usually at once involved, and the stomach contents escape into the general abdominal cavity unchecked by adhesions. A callous ulcer, on the other hand, has its base covered with granulations. Its base, but more especially its margin, shows the result of long-standing reactive inflammation, and the stomach walls are correspondingly thickened. Hence perforation in this type of ulcer is much more unusual than in the acute round ulcer, and when perforation does occur, the base of the ulcer is frequently adherent to some neighboring organ, particularly the pancreas or the liver, or is so protected

by adhesions to the intestinal tract or the omentum, that general infection of the peritoneum rarely occurs at once. The formation of a

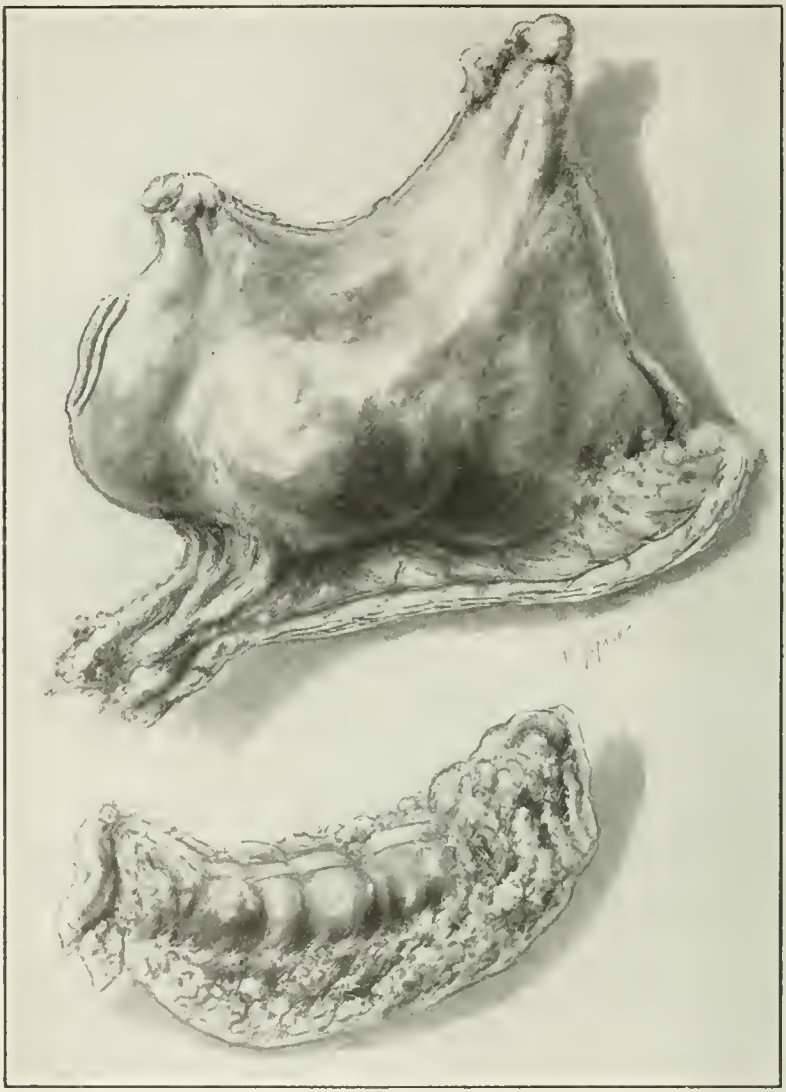

FIG. 30.—Above is Seen a Specimen Removed by Partial Gastrectomy: Callous Ulcer of the Anterior Wall of the Stomach. Division of Adhesions to the Mesocolon Damaged the Circulation of the Transverse Colon, a Section of Which Was Simultaneously Resected, and is Represented in the Lower Drawing. Patient Free from Symptoms One Year Later. See Also Fig. 31. From a Patient under the Junior Author's Care in the Episcopal Hospital.

subphrenic or subhepatic abscess, or an empyema of the lesser peritoneal cavity, very frequently in these cases precedes generalized

peritoneal infection, and renders possible the application of surgical measures in time to prevent the latter complication.

The erosion seem more apt than other forms of ulceration to give rise to sudden and overwhelming hemorrhage, appearing as the first symptom of gastric lesion, and at times leading to sudden death. The bleeding is in the nature of a general venous ooze. Usually a history of some recent acute constitutional infection (grippe, tonsillitis, bronitis, etc.) is obtainable in these cases. The punched out ulcer gives rise to acute and recurring hemorrhages by ulcerating into a blood vessel. The bleeding is as a rule safely checked by non-operative means, but operation is indicated to prevent a recurrence of this alarming feature. The callous ulcer is the variety which produces, more frequently than any other, those occult hemorrhages which induce the severe anemia not infrequently encountered in this disease.

In form, the punched out ulcer is generally well described by its usual name of "round ulcer," while the callous ulcer is more or less irregular in outline, sometimes appearing as if formed by the coalescence of several smaller ulcers, and usually having its long axis transverse to the long axis of the stomach. It is this form of ulcer which is chiefly productive of gastric distortions, such as pyloric stenosis and hourglass stomach.

Symptoms

Gastric ulcer is a chronic disease, with acute or subacute exacerbations; and as it is usually during one of these exacerbations that the patient seeks medical advice, the attention of physicians was for years focussed on the symptoms present at these times, namely *pain*, *vomiting*, *tenderness*. It is convenient, therefore, first to describe in some detail these definite symptoms and the two main complications of gastric ulcer (hemorrhage and perforation), and finally under the head of diagnosis (p. 92) to attempt a summary of what is at present known of the features of this many-sided disease.

Erosions and **ulcerations** seldom give rise to symptoms other than hemorrhage or perforation. They form the most acute class of gastric ulcers not only in the sense of their symptomatology, but also in their duration, since most of them heal very rapidly without having at any time been productive of symptoms of any kind.

The **typical gastric ulcer** is that form of the affection which of all others is characterized by *pain*. It should not be taken for granted, however, that no ulcer exists when pain is absent. As previously noted, an open ulcer may remain latent until its presence is announced

by hemorrhage or perforation. But the pain when it does occur is sufficiently characteristic to make it necessary for the physician to give it his careful attention. When the stomach is empty there is rarely any pain, but very soon, sometimes immediately after food is swallowed, a soreness or a sharp stabbing pain will arise at some well-defined spot in the epigastrium; and this soreness will persist, and probably grow continually worse, until the stomach is emptied, either by vomiting or by the discharge of its contents into the duodenum.

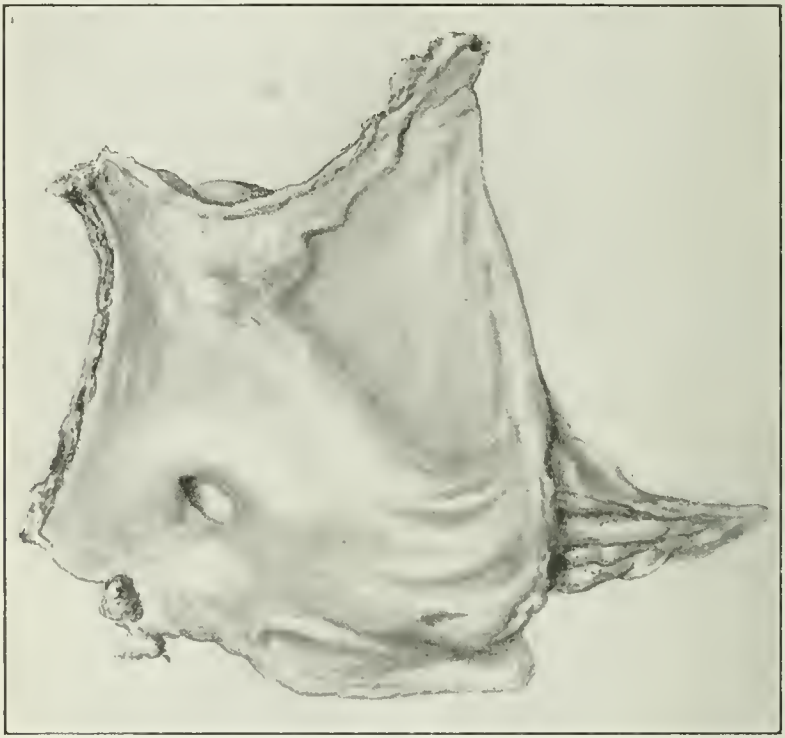

FIG. 31.—The Stomach Shown in Fig. 30 Has Been Cut Along the Lesser Curvature, and the Anterior Wall Has Been Turned Down Along the Greater Curvature as a Hinge, Exposing the Deep Crater of the Callous Ulcer Forming the Palpable Tumor in the Anterior Wall. (*Episcopal Hospital.*)

It is not possible to decide, from the site of the pain or from the interval after ingestion of food at which it first appears, in precisely what region of the stomach the ulcer is situated, nor to determine the nature of that ulcer, though it may be taken as a general rule that the longer the interval between ingestion of food and occurrence of pain the nearer the ulcer is to the outlet of the stomach.

The cause of the pain is still a matter of dispute. Although the nerve supply of the stomach is largely derived from the pneumogastric

nerve which carries both sensory and motor fibres, most observers agree that the stomach is devoid of common sensation such as that with which the skin is endowed. Lennander (1901) demonstrated that pain occurs on irritation of the parietal peritoneum, but that the viscera are insensitive unless their mesenteries are pulled upon; and this observation is frequently confirmed by surgeons who operate under local anesthesia. Hertz, in his Goulstonian Lectures (1911), explained visceral pain by tension on the walls of the viscera; thus in ulcer of the stomach peristalsis causes pain because the ulcer interrupts the normal peristaltic current, as can be seen on the fluoroscopic screen. Similarly, pyloric obstruction causes pain because the stomach is stimulated

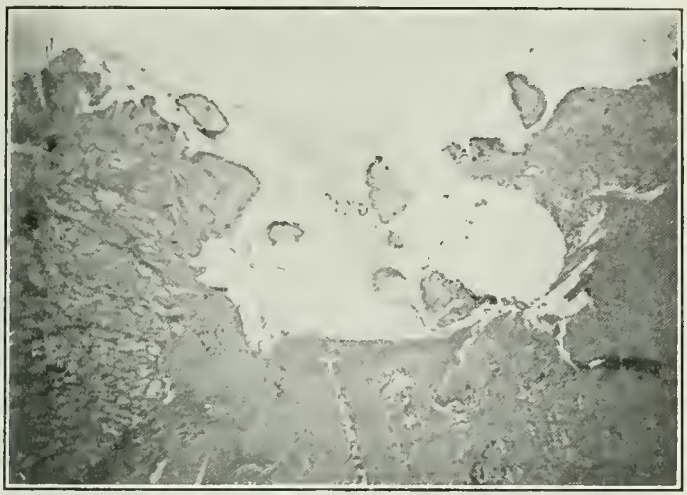

FIG. 32.—Microphotograph of a Chronic Gastric Ulcer, Showing the Overhanging Edge. (*Dr. Reiman, Lankenau Hospital.*)

to hyperperistalsis. Thus it is no longer believed that the acid gastric juice causes pain by mere contact with the gastric ulcer, but only because by coming into contact with the ulcer it stimulates irregular peristalsis. Where adhesions to the parietal peritoneum are present dragging upon them is another factor in the production of pain.

Accompanying the pain, and usually also present in the intervals between food, when pain is often absent, will be found a more or less well-defined and constantly located area of *tenderness* to pressure. This is usually in the costal angle, a little to the right of the middle, and varies from one to six or seven centimeters in diameter. The fact that this area of tenderness does not vary with the position of the stomach is now generally acknowledged; hence its location is no indication of the site of the ulcer.

The pain is not infrequently *referred* to some other region as well as to the epigastrium, particularly to the left hypochondrium and neighborhood of the left scapula. But more frequently it is the tenderness that is referred, particularly to the dorsal region. Peculiarly characteristic in advanced cases is a tender spot to the left, more rarely to the right, of the last two thoracic vertebræ. Pain and tenderness which are referred are much less usual in these open ulcers than in those which have formed adhesions in the course of their cicatrization and contraction.

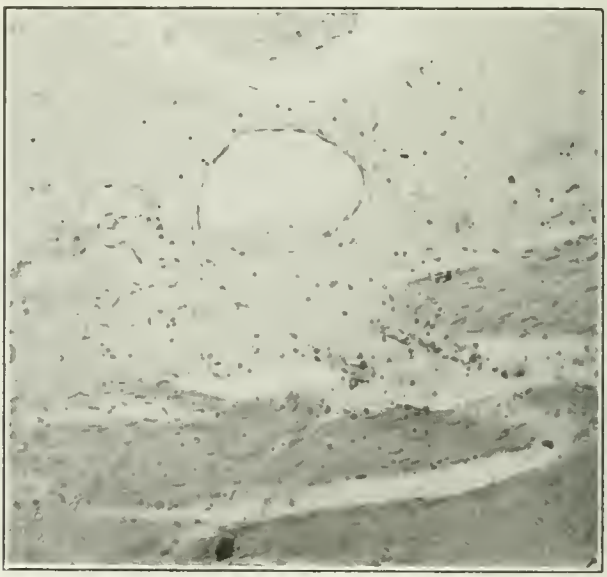

FIG. 33.—Microphotograph, Showing Edema of the Muscularis Occurring During an Acute Exacerbation of a Chronic Gastric Ulcer. (*Dr. Reiman, Lankenau Hospital.*)

Vomiting, next to pain, is the most constant symptom of gastric ulcer. In open ulcers vomiting is often induced by the pain which the ingested food causes, and some patients will voluntarily produce emesis, by gagging themselves, merely to be relieved of their gastric distress. The act of vomiting may occur spontaneously soon after a meal is taken, but when it is a constant feature of the disease, it does not usually occur until at least half an hour or an hour after eating. The vomited matter is notably acid and malodorous, and is often streaked with blood. The vomiting usually is not repeated until after the next meal. Many patients will have nausea but no vomiting.

Eructations and *heartburn* are very frequent symptoms of gastric

ulcer, but their significance usually is overlooked, and it is not until they are succeeded by actual vomiting and distressing pain that an anatomical cause is sought for.

Hematemesis may occur at any time that the ulcer invades a blood vessel, independently of the post-prandial vomiting. Profuse and prostrating hemorrhage may occur as the first symptom of gastric disease, and is then often due to erosions, being a venous flow whose precise origin rarely is discoverable either at operation or autopsy. The possibility of there being varicosities in the stomach or esophagus should not be overlooked. When the hemorrhage is more moderate in amount, but repeated at intervals of days, weeks or months, it is especially significant of the acute round ulcer. Any sudden increase in arterial tension may give rise to such a hemorrhage. The process of digestion, with the accompanying increased activity and hyperemia of the stomach, is naturally the most frequent cause; but sudden excitement, straining at stool, and vomiting, act in the same way. Hematemesis is rare at night when the stomach as well as the whole body is at rest. Vomiting of arterial blood is present, according to most observers, in from 30 to 50 per cent. of all cases of gastric ulcer; and minute traces of blood usually can be found in the vomitus in about four-fifths of the cases. Although it is the round open ulcer that is particularly characterized by vomiting of arterial blood, yet this symptom may be present in the callous ulcer. In the latter form, however, the blood is usually much less in quantity, and is more frequently clotted before it is vomited. Hemorrhage, as well as vomiting are later signs of gastric ulcer than is pain.

Hemorrhage into the stomach in cases of gastric ulcer does not always produce immediate vomiting. The blood may lie in the stomach and before vomiting occurs may become clotted or intimately mixed with food which has been ingested subsequent to the occurrence of the bleeding. Or the blood may be passed into the intestines and be finally evacuated in the stools, in which case it is much more likely to be overlooked.

A consideration of hemorrhage leads naturally to a discussion of the *anemia* which has for so long been regarded as a characteristic feature of the open gastric ulcer. It is still an undecided question whether the typical anemia is a cause or a result of the disease; but it is at any rate certain that it is a fairly constant feature, and opinion at present is inclined to consider both the anemia and the gastric ulcer as produced by the same cause—toxemia. Particularly in young females is this anemia noticed. A growing girl or a young

woman who is anemic and has indigestion should be very carefully examined for evidence of gastric ulcer. The anemia is usually of the chlorotic type—that is to say, there is a decrease in the number of the red blood cells, and a still greater decrease in the amount of hemoglobin present; the cellular elements being more quickly replaced after hemorrhage than is the hemoglobin. Inanition due to the vomiting and to the impaired digestive powers of the individual, as well as the recurring hemorrhage from the ulcer itself, are important factors in maintaining an anemia which has once developed.

Callous Ulcer.—This form of ulcer is characterized less by acute pain and hematemesis than by persistent indigestion, melena, which is often overlooked, and symptoms of obstruction of the pylorus. The vomitus may occasionally be streaked with blood, but the hemorrhage is rarely large in amount, except when fatal from perforation of some large vessel. The pain is not so marked as in open ulcer, the area of tenderness is not so well defined, and vomiting does not occur so soon after the ingestion of food. The pain is much more apt to be referred to some other region of the body, particularly to the left scapular region, and is largely due to pulling upon adhesions. Perforation is rare, and when it occurs is in the immense majority of cases either subacute or chronic in type, much more frequently producing perigastric or subphrenic abscess than immediate generalized peritonitis. A palpable mass is not unusual in a patient with a callous ulcer, and sometimes may closely simulate malignant disease (Figs. 30 and 31). The most characteristic features, however, are those of dilatation of the stomach, with food stasis, and regurgitant vomiting—subjects which will be considered in detail in a subsequent chapter.

Hemorrhage in Ulcer of the Stomach

As a general statement it may be said that any of the pathological varieties of ulcer may be the origin of any form of hemorrhage—of a sudden and fatal flow of blood, which kills in a few minutes; of repeated hemorrhages alarming in quantity but not immediately fatal; or of occult hemorrhages only to be detected at times by persistent and painstaking microscopical examination of the vomitus and the feces. But as a rule those hemorrhages which occur as the first symptom of ulcer, and which quickly kill the patient by their mere quantity, are found to have their origin in erosions of the mucous membrane, and not from any well defined ulcer. This is the rule, and if we could know that such a form of ulceration existed, we should expect any

hemorrhage which might occur to be profuse and overwhelming in quantity and to be quickly fatal.

Fortunately innumerable individuals, during some acute general infection, may develop erosions which will heal without causing any symptoms whatever, certainly without any hemorrhages. The following case is cited as an example of such hemorrhages, eventually proving fatal.

Repeated Hematemesis; Direct Blood Transfusion; Temporary Improvement; Operation; Recurrent Hemorrhages, and Death

F. H., male, aged twenty-seven years, admitted to the Episcopal Hospital Sept. 1, 1914. Eight years previously he had had an attack of severe abdominal pain with vomiting, which confined him to bed for almost a week. Since this attack he had enjoyed excellent health, with never a symptom of indigestion, until the last week, during which period he had suffered from distention after meals. The day before admission he vomited 1000 cc. of bright red blood, and on the morning of admission he vomited 500 cc. of blood. The feces had been perfectly black for the past few days.

On admission the patient was anemic, pale and exceedingly restless, with a profuse clammy sweat over the entire body. Respirations were rapid and shallow; pulse 127, of low tension and volume. Aside from exaggerated peristalsis the abdomen was entirely negative. R.B.C., 2,840,000; Hb. 42%; color index 0.75.

Two days after admission the patient vomited 1000 cc. or more of bright red blood, together with a few dark clots. Morphin had been given in sufficient quantities to check peristalsis. The patient complained of intense thirst. His extremities were cold, and the body temperature was subnormal. The pulse was soft, small, rapid, and at times could not be detected. R.B.C., 1,820,000, Hb. 30 per cent., color index 0.83. Direct transfusion was now done (Drs. J. W. Moore and J. P. Jones). Within a short time color returned to the blanched lips and the luster to the eyes. At the end of the transfusion which consumed 12 minutes, the pulse was full and bounding, the R.B.C., 3,110,000; the Hb., 60 per cent. and the color index 0.96.

Improvement was now rapid, and as no more hemorrhages had occurred, on Sept. 11, nine days later, the hemoglobin being 62 per cent., Dr. Ashhurst did a posterior no-loop gastro-jejunostomy, at the same time inverting a small area on the anterior gastric wall, 4 cm. from the lesser curvature, and midway between pylorus and cardia. This area was covered by a patch of recent lymph, and was thought to be over an open ulcer, especially as it lay directly in the course of a large vein running up to the lesser curvature.

The patient did remarkably well until one week after operation, when, with only a few minutes warning, he vomited a pus-basin full of bright red blood. This was the first time he had vomited since the transfusion over two weeks previously. He continued to vomit blood the entire day, though morphin, horse serum, etc., were pushed to the limit. A donor being at last secured, a second blood transfusion was attempted, but the patient succumbed thirty minutes after its termination.

At autopsy numerous ulcerations, varying in size from a pinhead to a pea, were found, involving only the mucous membrane of the stomach, in the region of the pylorus, and mostly on its posterior wall. No single lesion could be identified as the source of the hemorrhage. The anastomosis was intact.

Bleeding, more moderate in amount, though perhaps still alarming at times, but which ceases before the patient is entirely prostrated, and again recurs in similar amounts at irregular intervals of a few

weeks or months, is the form of hemorrhage which is specially characteristic of the round open ulcer. In such cases the ulceration in its progress opens some medium sized arterial twig, and free hemorrhage occurs until retraction and contraction of the opened vessel allow its mouth to be closed by clotting. Venous twigs are more rarely opened by these ulcers, possibly because the current of blood in them is slower, the tension less, and clotting occurs before or as soon as the vessel is opened. When repeated vomitings are merely streaked with blood, the hemorrhage has probably arisen in some such way as this. The callous ulcer has as its characteristic form of bleeding the "occult" hemorrhage already mentioned. The area of the stomach affected is in these cases anemic, being very largely formed of scar tissue, and any bleeding that occurs usually comes from some abrasion of the stomach wall consequent upon the stenosis present. But if, in this form of ulceration, a blood vessel of any size is perforated, the very thickness and rigidity of the margins of the ulcer, which were, before, the patient's protection against hemorrhage and perforation, promote now his destruction, since they absolutely prevent retraction and contraction of the opened vessel, and bleeding continues until the patient is dead. It is by such a process as this that the splenic or hepatic artery or one of their branches occasionally is perforated, the chronic ulcer having long since contracted adhesions to the pancreas or its surrounding structures, and the artery being fixed in a vise of cicatricial connective tissue.

Hemorrhage from an ulcer of the stomach is not always manifested by vomiting. In some cases of profuse hemorrhage the patient suddenly turns pale, becomes giddy, gasps for breath, is exceedingly thirsty; his pulse becomes feeble and rapid, his stomach feels full and warm, and syncope may be followed by death without further warning. If the bleeding be less profuse, and still no vomiting occur, life may be prolonged for a couple of days, the patient presenting, after the symptoms of internal hemorrhage, those of mild sepsis, with fever and delirium. Black spots may float before the eyes, or the amblyopia may be complete.

The more profuse the hemorrhage, the more apt it is to be due to ulcer, and not to carcinoma or to portal congestion or gastritis. In these latter conditions the amount of blood lost is rarely more than 50 to 75 cc., and usually clots before being vomited; but 500 cc. or more is not infrequently lost in cases of ulcer. After a hemorrhage of even less amount, the patient may present the signs of secondary anemia for some weeks. Dyspnea may occur on the least exertion;

the extremities may remain cold and clammy; tinnitus aurium may be an annoying feature; restlessness or even delirium may disturb the sleep. Irregular fever may persist in some patients for many weeks. This symptom, although always arousing suspicions of perigastric abscess, may be due to mild sepsis from a sloughing ulcer, which even a successful gastro-enterostomy cannot relieve at once, or may possibly be merely an evidence of the severe secondary anemia.

Perforation of Gastric Ulcers

This serious complication, which is said to occur in about four per cent. of all cases,[1] and to constitute eighty per cent. of the deaths due to the disease, arises from various and rather ill-defined causes. As in the case of hemorrhage, so also a perforation may be inaugurated either by physical means, or by the pathological processes of ulceration or sloughing. When an acute ulcer is subjected to sudden strain, as in vomiting, or is abraded by ingested food, its base may give way, and the peritoneal cavity be opened. Chronic ulcers are not so apt to be affected in this manner, partly because of the thickness of their bases and margins, but more especially because perigastritis usually has existed for some time, and as a consequence the general peritoneal cavity is protected by adhesions. Adhesions are said to be present in about two-fifths of the cases of perforation. When, in an acute ulcer, the perforation arises from the physical causes just mentioned, the solution of continuity is frequently slit-like in character; but when due to the extension of the ulcerating process, or to sloughing, the perforation is more or less circular. It is generally found that the larger perforations are due to the separation of a slough, and that those produced by progressing ulceration are minute in size, and give rise to symptoms less acute than the other varieties of perforation.

Perforations vary by actual measurement from those which can be classed merely as pin-point to those which will admit two or three fingers and measure 5 to 8 cm. in diameter. The average perforation however, does not exceed 3 to 5 mm. in diameter.

As a rule there is not more than one perforation present; but in about 20 per cent. of cases two or more have been found. Hence it is always well to search the gastric surface thoroughly, and in cases of doubt to establish free drainage of suspicious regions.

[1] This figure should be much higher if only those patients with gastric ulcer who seek surgical relief are considered; in a series of 44 operations for ulcer of the stomach at the Lankenau Hospital, 9 perforations (7 acute, 2 subacute) were encountered (20.4 per cent.).

About 70 per cent. of perforations are on the anterior wall of the stomach; and those situated on the anterior wall toward the pyloric end and near the lesser curvature form about 80 per cent. of the total. About 18 per cent. occur on the posterior wall; while the fundus and cardia are very rarely the seat of perforation. The anterior wall of the stomach is exposed to the general peritoneal cavity, is subject to a greater degree of dilatation and contraction than is the posterior, and is also more exposed to external trauma acting through the abdominal walls. The posterior wall is placed in contact with the relatively rigid and immovable spinal column, within the limited confines of the lesser peritoneal cavity, and in close relation with the pancreas, duodenum, and Spigelian lobe of the liver. Being thus protected, ulcers on the posterior gastric surface are prone to induce perigastric adhesions as soon as any peritoneal irritation is developed, and being so reinforced are neither so apt to perforate, nor to produce diffuse peritonitis in the rare event of their actual perforation, as are those placed in less well-protected situations. The location of gastric perforations is well shown in Fig. 34.

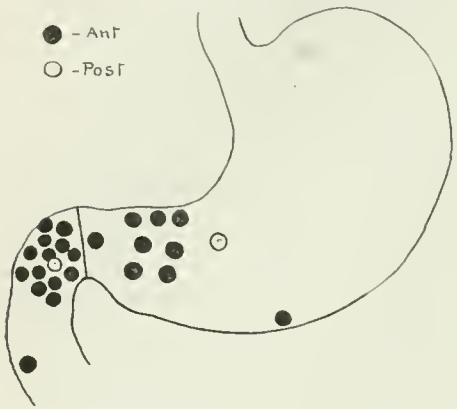

FIG. 34.—Diagram of Sites of 25 Perforated Gastric and Duodenal Ulcers Operated on by the Senior Author at the Lankenau Hospital. Perforations on the Posterior Wall are Indicated by a Dotted Outline.

Perforation of gastric ulcers has been well described as *acute*, *subacute*, or *chronic* in character. These terms refer not so much to the symptoms produced, as to the pathological course of the peritonitis caused by the perforation. An ulcer which perforates acutely is one such as those on the anterior wall which bursts through into the peritoneal cavity by sloughing or from the effect of physical forces, without having previously set up by contiguity a limited plastic peritonitis sufficient to protect, for a time at least, the general peritoneum from invasion. An ulcer which perforates subacutely is one whose base is gradually ulcerated through, so that perigastritis with its premonitory symptoms precedes the actual solution of continuity; and so that when this solution of continuity occurs, there is either a spreading plastic peritonitis already inaugurated, or adhesions are present which are more or less capable of limiting the outflow of the gastric contents.

By the term chronic perforation is inidcated that process which ensues when the base of an ulcer is adherent to some neighboring organ, so that scarcely any additional symptoms are produced at the actual moment when the gastric wall ceases to form the floor of the ulcer, and its place is taken by pancreatic or hepatic tissue, or by firm fibrinoplastic material—so that, in short, the symptoms which first call attention to the changed condition are not those of perforation nor peritonitis, but of sepsis, induced by slow absorption from some variety of perigastric or subphrenic abscess.

Subphrenic abscess (Chapter XV) has as one of its most frequent causes gastric ulcer. The term subphrenic abscess, so indiscriminately used is in many instances a misnomer, since the purulent collection is used, frequently subhepatic or retrocolic in location, and bears no direct relation to the diaphragm.

Symptoms.—The initial symptoms of perforation of any portion of the digestive tract bear a family resemblance to each other, and it is mainly by attention to the previous history of the case, and to certain rather ill-defined differential points, that a decision can be reached as to the particular part affected. Premonitory symptoms are not unusual, as pointed out by Robson and Moynihan. Recently one of us (Ashhurst) examined a woman at noon, who for a couple of days had been complaining of a stitch in the side—all her symptoms being referred to the left lower thorax and flank: examination was negative. Three hours later she developed symptoms of acute perforation, the ulcer being found at operation along the lesser curvature on the anterior wall near the cardia.

Sudden, severe, burning *pain* is nearly invariably the first symptom. It is usually localized in the epigastric or umbilical region, showing no tendency to shoot from one portion of the belly to another, nor to be referred to the hypochondriac or scapular regions. The pain is frequently so severe as to compel the patient to cry out; it affects him like a cramp: he doubles up his thighs toward his abdomen, and bows his body to his thighs, pressing his hands into his belly. Any one who has watched even one of these patients will never forget the sight. This agonizing pain may persist for fifteen or thirty minutes; seldom is it succeeded by symptoms of shock. Shock, recognized by the anxious cast of countenance, the cold and clammy surface, the pallor, the guarded breathing, and the quickening, feeble pulse occurs only when, through a perforation of extraordinary size, an overwhelming amount of highly toxic material is suddenly poured forth into a normal peritoneal cavity. It is due then to the toxemia produced by

sudden absorption from the peritoneal cavity of toxins and metabolic poisons in the effused gastric contents.

Usually, when first seen by the physician, the most acute symptoms have already passed, and the patient lies immobile on one side, or on his back with his legs drawn up, carefully guarding the abdomen.

After the first onset of pain, the patient often (in from fifty to sixty per cent. of cases) vomits, but this act is not commonly repeated, thus giving us one important point of distinction between perforation and obstruction or strangulation of the bowel. *Vomiting* is a serious feature, since it forces the stomach contents out not only by way of the esophagus, but also through the perforation into the peritoneal cavity. If the stomach be empty at the time perforation occurs, the nausea will produce retching only, and at most a little fluid, occasionally blood-stained, will be vomited.

Marked *rigidity* of the abdominal wall appears almost immediately, and its "board-like" character is especially characteristic of frank perforation; such board-like rigidity is seen also in gross perforations of the appendix, intestines, or gall-bladder; but not in peritonitis of slower onset not caused by gross perforations. If the infection be overwhelming, however, rigidity may never appear, but the patient will sink under the lethal influence of the toxic peritonitis, without an effort at repair of the lesion by plastic exudation. *Tenderness* arises at the same time as rigidity. The patient will no longer feel the pain as a cramp-like affection; he will draw up his thighs so as to relax the abdominal muscles, and will protect his belly from the slightest pressure. The tenderness thus developed may persist after extensive peritoneal involvement with its consequent tympany has rendered rigidity inappreciable. Of the two symptoms, however, rigidity is the more positive. *Distention* and *tympany* develop only with the progressive course of the peritonitis, and thus are late signs of perforation, replacing the original very characteristic rigidity only toward the tenth or eleventh hour.

Thirst is a very frequent symptom of gastro-intestinal perforations. Although nausea be present, and even if the initial vomiting be repeated, the patient will be very apt to drink water time and again, in the vain effort to relieve his thirst. During the height of the attack the *urine* is scanty or altogether suppressed.

Emphysema of the subcutaneous tissues, a very unusual and a very late symptom, is said to have been first noted in a case of gastric perforation by Demarquay (1866).

Immediately after perforation of any portion of the gastro-intestinal

tract, the *temperature* is apt to fall. We attach considerable importance to this symptom, and believe that if the temperature were taken without fail immediately after the initial pain appeared, it would be found subnormal with greater regularity than the statements of some writers would lead one to believe.

Accompanying or closely following the fall of temperature, there is a quickening of the *pulse*; and if operation be not undertaken promptly, the local tenderness and rigidity will spread over the abdomen. We have seen a very few cases in which the pulse was full and strong, and its rate slow. Whether this has any significance we do not know. (See p. 596 and p. 643.)

Diagnosis.—Acute, overwhelming pain, vomiting, fall of temperature, rise of pulse, and peritoneal reaction, i.e., *early* rigidity, followed from ten to eleven hours later by distention—these are the symptoms of perforation into the peritoneal cavity in general; and, as was remarked in the beginning, it remains to determine in the presence of these, what portion of the gastro-intestinal tube is affected.

The surgeon's thoughts naturally turn to gastric or duodenal ulcer as the cause of the perforation, and ninety-nine times out of one hundred he would be correct, even in the absence of a history of gastric disease. Such history usually can be elicited in from 50 to 75 per cent. of patients.

The diagnosis in typical cases is easily made by the hospital interne of average intelligence, from the text-book descriptions. Yet if the cases are seen late, it is not always easy to make the correct diagnosis. Thus in a series of 59 cases from the Cook County Hospital, studied by Scully (1918), only 5 of which came to operation in less than 5 hours after onset of symptoms, a correct diagnosis was made only in 27 cases (45.7 per cent). The other diagnoses were:

Acute appendicitis	9
Acute cholecystitis	3
"Acute abdomen"	17
Ileus	2
Liver abscess	1
Correct	27
	59

In an adult man, duodenal ulcers are more apt to be the cause of perforation than are gastric, and where no history is obtainable, that portion of the digestive tract should be explored first in such patients.

Of course if there is a clear history of preceding gastric symptoms, it is difficult for no one to make the diagnosis of gastric or duodenal perforation; but in the absence of such history there are no pathognomonic symptoms by which we can certainly distinguish between perforation occurring in the upper and that occurring in the lower portions of the abdomen. Those portions of the gastro-intestinal tract which most frequently perforate are the appendix, the duodenum, the stomach and the ileum, and the frequency of perforation is probably in the order named. But perforation of the appendix is rare compared to the development of peritonitis from appendicitis without macroscopic perforation, and even when it does perforate into the general peritoneal cavity, the symptoms produced are not of so alarming and prostrating a nature as when some portion of the digestive tube with a larger calibre is perforated, even when a large perforation occurs at the base of the appendix. Perforations of the ileum are rare, except during typhoid fever; and the occurrence of symptoms of peritoneal perforation during typhoid fever naturally suggest the ileum as the part affected. Typhoid ulcers of the stomach are extremely rare.

In diagnosticating perforations of the upper abdomen, when the clinical history is negative, we must rely chiefly upon the location of the initial pain, upon the excessive board-like rigidity (rarely seen in appendicitis even with diffuse peritonitis), and upon the directions in which peritoneal involvement proceeds. Although perforations in other portions of the abdomen sometimes produce epigastric pain, it is rare for gastric perforations to cause other than epigastric pain. Then, too, in gastric and duodenal perforations, the general peritoneal cavity is more quickly involved than in perforative lesions of the lower abdomen. In the latter it is the pelvis that is usually first involved by extension, and the umbilical and epigastric regions do not become affected until later; whereas when the perforation is in the stomach or duodenum, these regions are immediately affected, and it is only by gravitation of liquids that the lower portions of the abdomen are involved. In the majority of gastric perforations the transverse colon and the great omentum protect the hypogastric region and the pelvis from invasion, and as the splenic flexure of the colon is higher than the hepatic, any fluid extravasated above it tends to flow toward the right. These facts, as well as the anatomical relations of the ascending mesocolon, tend to make fluids from perforations in the upper abdomen collect first in the right kidney pouch, and then in the right iliac fossa following the paracolic groove. When a patient presents such symptoms as these when seen for the first time, the resemblance to appendicu-

lar abscess is very close, and the diagnosis of gastric perforation is scarcely ever made before operation, especially where no history of previous gastric disease can be elicited.

In some instances, when the existence of a gastric ulcer is known, the onset of a subacute perforation may be recognized by premonitory symptoms for several days beforehand. Fleeting pain of a stab-like character, or merely vague feelings of increased gastric distress may be present; and cases are known where deep breathing or sudden turning of the body have caused exacerbations of the pain by a sudden pull on newly formed adhesions. Robson and Moynihan mention a patient who said that it hurt her to bend, as her side felt stiff. In cases such as these the moment of actual perforation is not attended by such violent pain nor by such marked collapse as when the perforation is acute, and a temporary lull may occur, during which the symptoms abate, and a perigastric abscess forms. Unless promptly relieved by operation such patients will perish from sepsis or from subsequent generalized peritonitis.

The symptoms of chronic perforations of the stomach are those of perigastric and subphrenic abscess. Strict attention to the clinical history of the patient, with a painstaking and if need be oft-repeated physical examination will enable the diagnosis to be made in the majority of cases. Evidences of sepsis, with progressive emaciation, hectic fever, chills and sweats, and, above all, persistent tenderness to pressure will be the surgeon's best guide. Further consideration is given this subject in Chapter XV.

Differential Diagnosis.—Although, with a clear history and characteristic symptoms, such as those already detailed, the diagnosis of gastric perforation may be considered an easy problem, yet in not a few instances mistakes have been made by capable surgeons. Even with symptoms so typical as to leave no reasonable room for doubt, abdomens have been opened, and yet no lesion whatever has been found, and the patients have gone on to satisfactory recovery. And in a somewhat less limited number of cases some lesion other than gastric perforation has been discovered either at operation or autopsy. The importance of attentively considering the differential diagnosis of this complication is therefore very evident.

Perforation of duodenal ulcer is the condition which in every respect most closely resembles the perforation of a gastric ulcer. Indeed, so close is the resemblance that differentiation from symptoms alone is usually impossible, and the surgeon must rely on the age, sex, and previous history of the patient in drawing his conclusions. Since

immediate operation is indicated in either case, the distinction in this instance is rather of academic than practical importance.

Though rare, it is not an unheard of thing for peritonitis to arise in gastric disease without any actual perforation. McCosh (1900) recorded the case of a patient in whom strangulation of a gastric polyp produced *gangrenous gastritis*, with the formation of an abscess which gravitated to the right iliac fossa, and later caused death from general peritonitis. Harte (1901) observed a case in which a *septic gastritis*, arising during an attack of acute nephritis, with uremia, caused all the symptoms of a gastric or duodenal perforation, and in which death ensued from generalizd peritonitis, no macroscopic perforation being discoverable at autopsy.

Acute appendicitis differs in several respects in the symptoms it usually presents, and as a rule need not be confused with a gastric perforation, especially if the latter be of the acute variety. Many cases of subacute perforation of the stomach resemble suppurative appendicitis very closely, and are frequently not to be distinguished until after the abdomen has been opened. In acute appendicitis the initial pain is diffuse, colicky, and wave-like in character; at a later period it settles into the right iliac fossa. There is little if any collapse; fall of temperature is rare; abdominal rigidity is well localized to the region of the appendix, and general peritoneal invasion is often delayed for one or two days, a palpable mass meantime forming in the right iliac fossa. The pain in acute gastric perforation is overwhelming, and though local at first, very quickly becomes general; shock may be present, though rare, the temperature may fall, and the patient may appear in imminent danger of death; there is widespread, board-like rigidity at first, but as reaction commences, evidences of general peritonitis are found, at a much earlier period than is the case in appendicitis. Some cases of appendicitis, however, so closely resemble a gastric or duodenal perforation, that differentiation is impossible. Especially is this true in the case of large perforations at the base of the appendix, and sometimes in the case of a ruptured appendicular abscess.

Ruptured extrauterine pregnancy resembles gastric perforation in the agonizing pain. But the previous histories are different; the location of the pain is not the same; evidences of internal hemorrhage frequently persist, and overshadow the rather tardy development of peritonitis; and a vaginal examination may detect an exquisitely tender tumor in the pelvis. Occasionally there is profound collapse.

Acute intestinal obstruction is not characterized by symptoms of

peritonitis until strangulation occurs. Vomiting is persistent, becoming stercoraceous in time; rigidity is not pronounced, and relief from the pain may even be obtained by pressure and massage of the abdomen at a time long after diffuse peritonitis would have arisen were the case one of gastric perforation. Obstipation usually is absolute; collapse is wanting unless perforation occurs above the strangulated area, and the temperature remains normal or subnormal until the advent of peritonitis. The previous history of the patient may reveal the cause of the obstruction in long-standing peritoneal adhesions, or physical examination may detect a strangulated hernia or an ill-defined abdominal tumor—possibly a volvulus, an intussusception, or a pelvic tumor with twisted pedicle.

Gall-stone colic and *acute cholecystitis* are usually sufficiently distinguished by their clinical history, the location of their physical signs, with the slower development of peritonitis.

Acute hemorrhagic pancreatitis resembles gastric perforation in the intensity of the pain but no history of gastric ulcer is as a rule obtainable, and the typical subjects of pancreatitis are obese, alcoholic individuals of middle life while gastric perforations are comparatively rare after the age of 30 years.[1] There may be palpable, in the region of the pancreas, a deep seated tumor, which does not move with respiration, and which may reveal an indistinct sense of fluctuation. These features somewhat resemble those which arise in cases of perigastric abscess or empyema of the bursa omentalis, due to subacute or chronic perforations of gastric ulcers; and though some assistance in making the diagnosis may be obtainable by the possible occurrence, in pancreatitis, of jaundice, fatty diarrhea, and glycosuria, yet in many cases differentiation before opening the abdomen is impossible.

Mesenteric thrombosis is another affection which is sometimes confused with perforation of gastric ulcers. It is, however, a comparatively rare occurrence; and is not characterized by abrupt onset, nor by early peritonitis. Indeed, when the thrombosis is arterial, there are no well-recognized symptoms by which a diagnosis can be made, the affected bowel becoming the seat of dry gangrene. If venous obstruction arises, the symptoms are more acute: there are vague abdominal pains, continuous but paroxysmal; a little fever, possibly vomiting; sometimes bloody stools; and finally the evidence of peritonitis. But the course is much less acute than in gastric perforations, and all the symptoms less severe.

[1] The junior author has operated on a man 84 years of age with duodenal perforation.

Ptomain poisoning is characterized by a similar onset—acute abdominal pain, nausea, and vomiting, but often these are followed by collapse, which may be great, the temperature subnormal, and the skin cold and clammy. Diarrhea, moreover, is often a salient feature, and the vomiting is more persistent than in cases of gastric perforation. Although the physical signs—tenderness, rigidity, followed by distention—are the same in both affections, distention occurs much earlier in ptomain poisoning than when due to peritonitis from gastric perforation. In ptomain poisoning, also, a history of the ingestion of suspected food stuffs can usually be obtained; and a period of incubation (varying from twelve to thirty-six or forty-eight hours) between the ingestion of the poison and the development of symptoms will usually be found to have elapsed. During this period of incubation the patient may have been seemingly well, or there may have been fleeting pains in the abdomen, and more or less *malaise*. This distress, however, is intestinal, not gastric; and the history does not in the least resemble that of gastric ulcer.

Skin Diseases.—Certain affections of the skin, whose pathology is still very obscure, are at times attended by gastro-intestinal crises. This is particularly true of those affections belonging to the erythema group, and while they are more common in children, in whom gastric perforations are extremely rare, they are not unknown among adults. The occurrence of purpura, angeio-neurotic edema, erythema, or urticaria, with recurring colic, and often albumen in the urine, are the symptoms most significant.

Gastric crises of tabes dorsalis should be kept in mind in atypical cases. Neglect of a complete physical examination has several times mislead a surgeon in these patients.

Diagnosis in Cases of Gastric Ulcer

It scarcely seems necessary, after the account of the affection just given, to dwell at any great length upon its diagnosis. Yet this is not always an easy matter, in spite of the succinctness with which the symptoms may be detailed. The *clinical history* of the patient is the feature of the disease which is most constant, and which must, in our judgment, take precedence over the physical examination, and over diagnosis by means of laboratory methods, and skiagraphy. Too little attention is commonly paid both by the family physician and the consultant to the importance of eliciting a clear and untrammeled account of the origin and progress of the malady from which the patient

suffers. In cases of doubt, the attendant should return to the charge again and again, and should endeavor, without putting upon the patient's tongue any false answers, to obtain from him such responses as will, when strung together in chronological order, reveal the natural course of the disease. Very many patients will have forgotten the earliest symptoms from which they suffered because they were ignorant of their significance; and it may not be until they have been questioned two or three times that some event, such as slight hematemesis, seemingly trivial in itself, will rise again to their memory, and perhaps supply to the history of the disease the missing link which so long had been desired. The diagnostician never should forget that the presence of one or two positive signs is worth infinitely more than the absence of many others.

The features of most value in the clinical history of the patient have been tabulated under the following heads by C. Graham (1913):

1. *Periodicity of attacks* of "indigestion" is especially characteristic of gastric (pyloric and duodenal) ulcers. These patients suffer for several days or weeks at a time with stomach symptoms (flatulence, pain, belching, etc.) and then for an indefinite period (usually for weeks or months) are almost if not entirely free from any discomfort. These recurrent attacks show a special predilection for the spring and autumn of the year, and seem to be brought on sometimes by worry, overwork, or some intercurrent infection (coryza, grippe, bronchitis, etc.).

2. *Chronicity of the Disease.* The train of symptoms above noted usually has lasted for years before the distress becomes severe enough to demand relief.

3. *Pain*, which has already been described in detail, is the most constant symptom *during the attack.*

4. *Time* and *control of pain* are also very characteristic. Pain appears after eating; in duodenal ulcer it may appear so long after eating that it is regarded as "hunger pain," i.e., occurring *before the next meal.* Then the fact that this pain is controlled by more food, by alkalies, by position, or by rest (vacation) is one of its most notable features. Graham thinks that the nearer the ulcer is to the gastric outlet the longer is the interval between ingestion of food and development of pain. But this means of localizing an ulcer close to or distant from the pylorus is very unreliable.

The *physical examination* must be considered as merely confirmatory of the clinical history, as previously elicited; and any chemical tests of the stomach contents are of value only as corroborating the physical examination, as are also the results of Roentgenological study. Indeed

most Roentgenologists do not attempt to make any diagnosis from their examinations except after careful study of the clinical history.

Blood examination may further confirm the diagnosis by revealing a chronic anemia. Friedman (1914) attempted a differential diagnosis between pyloric and non-pyloric ulcer, duodenal ulcer, and chronic appendicitis, on the basis of the morphology of the blood, especially the differential leukocyte count. It is said that the presence of the normal leukocytosis during digestion favors the diagnosis of ulcer rather than carcinoma.

Differential Diagnosis.— There are few affections with which a case of uncomplicated gastric ulcer need be confused. An exception to this statement is *ulcer of the duodenum*, which in many respects may so closely resemble gastric ulcer as to be indistinguishable. Its symptomatology is considered in Chapter VIII.

Acute gastritis and *gastro-duodenal catarrh*, while possibly productive of the symptoms of pain, tenderness and vomiting, are affections which may almost always be traced to some recent indiscretion in diet. The vomiting does not recur with any regularity after meals, in the effort to relieve the stomach of the pain which is present in cases of ulcer, but is the result of nearly constant nausea, which is characteristic of the inflammatory nature of the affection. The tenderness, if any is present, is diffuse, not accurately localized, as is usual in cases of gastric ulcer; hematemesis occurs only in the most exceptional cases; and jaundice, which is extremely rare in simple gastric ulcer, is a frequent accompaniment of gastro-duodenal catarrh. Finally, abstinence from food, with other appropriate treatment, quickly relieves the inflammatory affection, while the symptoms of gastric ulcer persist, or if cured temporarily are prone to recur as soon as energetic treatment is discontinued.

Hemorrhage from the gastro-intestinal tract due to other causes than gastric ulcer may be extremely difficult at times to differentiate from the latter affection. Particularly of hematemesis is this true. When the blood is discharged from the bowel, although duodenal ulcer should certainly be considered, there are usually other symptoms which will aid the diagnosis. Enterorrhagia is sometimes the earliest symptom of typhoid fever; it is infrequent as an early symptom of malignant changes in the bowel; but in both of these, as in almost every other conceivable case of bleeding from the bowels, there is almost invariably soon developed some other symptom or chain of symptoms which at once makes clear the nature of the malady.

Far different is the case with gastrorrhagia and hematemesis.

It is natural to assume that a patient presenting these symptoms suffers from gastric ulcer; and when all other lesions have been excluded, the supposition seems justifiable. The confusion of hemoptysis with gastric hemorrhage is not usual, and the difference in the physical signs between the gastric and thoracic disease, as well as the frothy character of the expectorated blood, tend to make such confusion, if it ever arise, rather short-lived.

A cause of sudden profuse hemorrhage which, until recently, has not received adequate consideration, is the rupture of varicose veins of the stomach or of the lower portion of the esophagus. It is not improbable that some of the hemorrhages formerly attributed to

FIG. 35.—W. J. Taylor's Specimen Showing Rupture of a Varicose Gastric Vein Near the Cardia. Note also Gastro-jejunostomy Opening, and Pagenstecher Thread Hanging Loose, with Piece of Undigested Vegetable Fibre Attached.

erosions were in reality due to the rupture of varicose veins. Preble (1900) collected sixty cases of fatal gastro-intestinal hemorrhage due to cirrhosis of the liver. Out of these sixty cases the esophagus was examined in forty-two; and among these there were found esophageal varices in thirty-five cases, or 85 per cent. The source of the hemorrhage, Preble states, was recorded in 19 cases, occurring 16 times from rupture or ulceration of esophageal varices, twice from ulcers over gastric veins near the cardiac vein, and once from an erosion near the cardia. He thinks it probable that in many of the cases in which varices were present ruptures had occurred which were overlooked, as they were not tested by injection with air or fluid. "The veins of the cardiac end of the stomach are part of the portal system, while those of the esophagus are part of the systemic system. Here, as at the lower end of the intestinal tract, the two systems are connected by anastomosing branches

which, as a rule, according to Orth and Kundrat, are too small or too few to contribute much to the formation of a collateral circulation when the portal system is obstructed. But when this anastomosis is free, they become an important factor, and their dilatation may so completely compensate for the veins obstructed in the liver that the clinical course of the cirrhosis is altered and obscured" (Preble).

The blood in the lower esophageal veins is returned to the heart by way of the lower azygos minor vein, as well as through some of the bronchial veins; and, as Preble points out, being intrathoracic in situation, they "feel the negative pressure of inspiration, while at the same time the pressure in the portal system rises to fourteen or even eighteen millimetres of mercury. In this way the blood is literally aspirated from the coronary into the esophageal veins, and leads to their permanent dilatation."

In patients, therefore, where the anastomosis between the coronary and the esophageal veins is free, cirrhosis of the liver may not be accompanied by its usual symptoms, such as ascites, enlargement of the spleen and of the subcutaneous abdominal veins. Among the patients with cirrhosis whose records were examined by Preble, in whom varices were present and caused hemorrhage, one-third died from hemorrhage without any accompanying sign of the cirrhosis; in one-third hemorrhage was the first symptom, and others followed; and in the other third the hemorrhage was preceded by other symptoms.

The surgeon, therefore, should be very cautious about undertaking operations on the stomach in the case of a patient who presents symptoms merely of gastric hemorrhage, without other evidences of ulcer. If the abdomen of a patient with esophageal varices due to cirrhosis of the liver be opened, there will usually be found an increased amount of peritoneal fluid, and palpation of the liver may detect the true condition. Under such circumstances, probably the best course will be to suture the omentum to the parietal peritoneum, in the hope of relieving the esophageal veins of some of their pressure. Certainly no relief can be expected from a gastro-enterostomy. In cases of postoperative hemorrhage we believe with Dieulafoy that the condition is generally due to a mild form of sepsis, and that in fatal cases one or more erosions or exulcerations could be found in the gastric or intestinal mucosa.

A distinction between open ulcer of the stomach and *gastric carcinoma* is not usually difficult; but in certain cases of callous ulcer with much thickening and many perigastric adhesions, the diagnosis is often a matter of considerable importance as well as difficulty, since

the question of radical treatment is at once raised. The distinction is discussed under the heading of malignant pyloric obstruction (see page 263). The duration of the disease is the most important factor in differentiating a simple ulcer from a cancer; but it must not be forgotten that while carcinoma is typically abrupt in its onset, first manifesting itself after forty years of age, yet that it may be implanted upon ulcer, and that a patient with an ulcer of many years duration may have a carcinoma which has only recently developed, but whose course, unchecked, will be quickly fatal. The age of the patient, moreover, is no certain guide as to the presence of cancer. As is well known, the gastro-intestinal tract is affected with malignant growths in younger patients than is any other region of the body.

Cholelithiasis usually is sufficiently distinguished by the location of the symptoms and physical signs, as well as by the extreme irregularity of the attacks of gall-stone colic, which bear no relation whatever to the ingestion of food, and which recur with no persistence after each meal, as does the pain due to gastric ulcer. Moreover, even in the intervals between the attacks of biliary colic, the digestion is never in perfect order, though actual distress may be absent. (See p. 485.)

Chronic appendicitis may very closely mimic the symptoms of gastric and duodenal ulcer, and is in many patients a coincident disease, the appendicular focus of infection being indeed not infrequently the original cause of the gastric lesions. But patients with uncomplicated chronic appendicitis usually are younger than those with ulcer of the stomach, most of the latter being over 35 years of age. In appendicitis the dyspeptic symptoms bear no relation to the ingestion of food, and no relief is obtained by eating or by ingestion of alkalies. Usually one or more attacks have occurred of sufficient severity to have laid the patient up in bed for a week or ten days, and in most cases attentive physical examination will detect deep tenderness in the right iliac fossa. Patients with chronic appendicitis, moreover, do not give the history of intervals of entire freedom from digestive disturbances which is so characteristic of gastric and duodenal ulcers in their earlier stages; and owing to the adhesions which early form around the appendix, any unusual physical exertion is very apt to cause a recrudescence of symptoms in cases of chronic appendicitis.

Chronic Pancreatitis.—The diagnosis of this condition is considered at p. 67c

Prognosis in Cases of Gastric Ulcer

The prognosis in cases of ulcer of the stomach may be considered under three headings: (1) Untreated; (2) Medical Treatment; (3)

Surgical Treatment. It is, however, extremely difficult to obtain any accurate data as to the percentage of deaths and permanent cures under the first two headings; and even with surgical treatment we must acknowledge that it is still of too recent adoption for us to draw definite conclusions.

It is not likely that a patient with an ulcer which produces symptoms of any severity will go untreated. But it must be remembered that many ulcers are latent, and first announce their presence by copious hemorrhage or lethal perforation. Van Valzah and Nisbet, as long ago as 1900, were able to find scattered through the literature fifty-eight cases of latent ulcer, the first manifestation of which was perforation.

But there are many patients in whom the symptoms of the disease are subacute in character, and persist for years, untreated, until finally some acute complication occurs, killing them, or at least bringing them very near death; or if no acute complication arises, the patients pass into the class of chronic gastric ulcer, with pyloric obstruction, gastric dilatation, or other more serious affections. Brinton gave the mortality from all causes in gastric ulcer as 50 per cent.; Lebert considered 10 per cent. a fair estimate; while Robson and Moynihan came to the reasonable conclusion that under medical treatment the mortality of gastric ulcer is at least 20 per cent. This mortality they divide between hemorrhage, which kills 5 per cent., and perforation, which kills at least 15 per cent. of patients with ulcer of the stomach.

When, in addition to these figures, we consider the other complications to which patients with gastric ulcer are prone, we begin to realize what an alarmingly serious disease it is, and how unsatisfactory medical treatment must be in a great many cases. Among other complications to which gastric ulcer may give rise must be considered, in addition to perforation and hemorrhage, pyloric stenosis, with its long train of maladies due to dilated stomach; hourglass stomach, and other results of adhesions and distortions; the transition of ulcer into carcinoma (see page 114); subphrenic abscess; and even progressive pernicious anemia, which is dwelt upon in particular by Hemmeter.

That medical treatment may accomplish much in patients suffering from open ulcer no one can deny. The immediate mortality of the disease may probably be reduced to 10 per cent. by the best medical treatment. But the treatment must be methodical and energetic. No half-hearted measures will suffice. As Van Valzah and Nisbet say, "Expectant treatment is a great blunder, for simple ulcer in a vigorous adult has no 'innate tendency to heal,' and the grave accidents which are too often the heralds of coming death occur somewhat regardless of

the age, the constitution, and the general state of nutrition. The treatment must in every case be immediate, methodical, and sufficiently vigorous to be effective. A compromise may mean death or irreparable injury."

To consider at present only the prognosis of open ulcer of the stomach, postponing that of the callous or cicatrizing variety to another chapter; it seem to us that a rational point of view is the following: to adopt at first in every case energetic medical treatment. Under this, the acute pain, the tenderness, and the recurring hemorrhages of open ulcer can almost invariably be checked, and in a certain proportion of cases, smaller we believe than medical men as a rule admit, these patients will remain cured. Unfortunately medical men are not agreed on what is the proper form of non-operative treatment. Many hold to the classical treatment of rest in bed, and local rest for the stomach procured by nearly total abstinence from mouth feeding for at least one week, while the patient's strength is supposed to be supported by nutrient enemata (Von Leube). Others of the modern school, will be inclined to adopt the Lenhartz method, of which Hort's treatment (1910), is an advanced example. This may be summarized as follows: (1) For acute cases, with hemorrhage, subcutaneous injections of normal horse serum, and feeding by mouth at the very earliest opportunity with small dry meals, mainly of meat; (2) For chronic cases, full meat diet in an appropriate form from the start, with repeated oral doses, never on an empty stomach, of an antilytic serum specially prepared. The rationale of Hort's treatment is based on the modern conception of the gastric or duodenal lesion as merely a symptom of a blood infection, and not as was formerly taught a disease in itself. Hort claims that with this understanding of the affection, the absorption of adequate protein in suitable form cannot but increase the resistant powers of the body as a whole to the unknown determining cause of the disease. He points out, further, that protein food will utilize the extra amount of acidity in the stomach; and that by adding at the height of digestion a serum with a high antipepsin and antitrypsin content, some of the unsatisfied residue of peptic and tryptic bodies will combine therewith, and the ulcer thus be considerably shielded by the diversion of these bodies. Finally, he claims that the local action of this serum on the ulcer will be beneficial. This antilytic serum (normal serum with the inhibitory content artificially raised) is on the market; and the dose is from 30 to 40 cc. daily, in divided doses of 10 cc. each, always with or directly after food. He claims there is no danger of anaphylaxis if the serum is

given by mouth only. Sippy (1915) adopts a method of treatment which has for its object the prevention of the corrosive action of the gastric juice on the ulcers. He gives largely a milk diet, with frequent very large doses of antacids, sufficiently large to neutralize the acidity of the gastric juice, which is repeatedly examined. No food residues are allowed to remain in the stomach, and thus there is no excitant for the flow of gastric juice during the hours of sleep when antacids cannot be taken by mouth. The patient is kept in bed for an average period of four weeks, and subsequently continues the treatment in modified form for months.

Statistics which were quoted at length in the first edition of this book, and which so far as we can ascertain are still representative of the results of medical treatment, lead us to conclude that when the symptoms of open ulcer are not relieved after energetic medical treatment lasting for several weeks — probably six weeks should be the outside limit — or where the disease recurs after being temporarily checked, that then some more radical treatment is required. This treatment is operative. The choice of operation does not concern us here. It will be discussed fully under the head of treatment. What we desire at present to learn is the immediate mortality of surgical treatment, and the proportion of ultimate and enduring cures that we may expect.

We have seen above that under medical treatment the immediate mortality is from 10 to 20 per cent. The mortality of untreated cases may be estimated at from 20 to 50 per cent. The average death rate for gastro-enterostomy, which is the operation still most frequently employed in these conditions, is as low in the average at the present day as 5 per cent., and in the hands of those surgeons who do many of these operations is even lower. The following statistics have all been reported since the appearance of the first edition of this book (1909).

STATISTICS OF OPERATIONS FOR BENIGN DISEASES OF THE STOMACH AND DUODENUM

Operator	Date	Number of operations	Deaths	Mortality per cent.
Bidwell	1911	181	13	5.0
Coffey	1920	233	10	4 33
Deaver[1]	1920	673	36	5 3
v. Eiselsberg	1914	334	17	5 0
Enderlen	1914	52	4	7 7
Finney	1915	200	12	6.0
Galpern	1911	170	6	3 5
Kocher	1910	43	0	0 0
Krabbel and Geinitz	1914	108	7	4 1
Kümmel	1912	71	4	5 6
Küttner	1911	56	5	9.0
Mayo Clinic	1915-1919	3480	53	1 52
Mitchell, A. B.	1911	110	3	2.7
Monsarrat	1912	58	0	0.0
Moynihan	1920	835	12	1.43
Sherren	1914	200	6	3.0
Short	1911	41	0	0.0

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v. Eiselsberg: Lancet, 1914, ii, 296.
Enderlen: Fanlhaber and Redwitz, Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1914, xxviii, 150.
Finney: Surg. Gyn. and Obst., 1914, xviii, 273. Am. Jour. Med. Sc., 1915, ii, 469.
Galpern: Arch. f. klin. Chir., 1910–11, xciv, 870.
Kocher: Dentsch. Zeitschr. f. Chir., 1912, cxvi, 183.
Krabbel and Geinitz: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1914, xxvii, 859.
Kümmel: Deutsch. med. Woch., 1912, xxxviii, 395.
Küttner: Ther. d. Gegenwart, 1911, liii, 19.
Mayo Clinic: Reports of St. Mary's Hospital, Rochester, Minn., 1915–1919.
Mitchell: Annals of Surgery, 1911, liv, 806.
Monsarrat: Liverpool Med.-Chir. Jour., 1913, lxii, 364.
Sherren: Surg., Gyn., and Obst., 1914, xix, 564.
Short: Bristol Med.-Chir. Jour., 1911, xxix, 220.

[1] In the first edition of this work there were recorded (up to the year 1908) 91 operations of all kinds for benign diseases of the stomach with 8 deaths, a mortality of 8.7 per cent. During the years 1908–1920 (Jan. 1) inclusive the senior author did 582 operations of all kinds for such diseases, with 28 deaths, a mortality of 4.81 per cent. These figures may be seen in detail in the accompanying table. The entire period (1900–1920) comprises 673 operations for benign diseases of the stomach and duodenum, with 36 deaths, a mortality of 5.3 per cent.

OPERATIONS FOR BENIGN DISEASES OF THE STOMACH AND DUODENUM

Lankenau Hospital (1909–1920) Immediate Mortality	Number	Recovered	Died	Mortality per cent.
Posterior gastroenterostomy for				
Ulcer, duodenal	239	229	10	
Ulcer, duodenal, perforated	32	31	1	
Ulcer, gastric	62	59	3	
Ulcer, gastric, perforated	12	10	2	
Ulcer, pyloric	2	1	1	
Ulcer, marginal	1	1	0	
Stenosis, duodenal	1	1	0	
Stenosis, pyloric	2	1	1	
Adhesions, pyloric	3	3	0	
Obstruction, pyloric	3	3	0	
Total	357	339	18	5.0
Partial gastrectomy for				
Ulcer, duodenal	6	6	0	
Ulcer, duodenal and gastric	1	1	0	
Ulcer, duodenal, perforated	4	4	0	
Ulcer, gastric	25	22	3	
Obstruction, pyloric	2	2	0	
Total	38	35	3	3.8
Pylorectomy for				
Ulcer, duodenal	57	54	3	
Ulcer, duodenal, perforated	1	1	0	
Ulcer, gastric	1	1	0	
Fistula, duodenal	1	1	0	
Total	60	57	3	4.6
Excision				
Ulcer, duodenal	49	48	1	
Ulcer, gastric	22	22	0	
Total	71	70	1	1.4
Resection, circular, stomach				
Ulcer, gastric	16	15	1	6.0
Jejunojejunostomy				
Obstruction, duodenal	1	1		
Duodenoduodenostomy				
Vicious circle	1	1		
Enteroenterostomy				
Adhesions, abdominal	1	1		
Roux-Y for				
Vicious circle	1	0		
Ulcer, marginal	1	1	1	
Total	2	1	1	50.0
Anterior gastroenterostomy				
Ulcer, marginal	1	1		
Gastrostomy for				
Stricture, esophagus	2	2		
Ulcer, marginal	1	1		
Total	3	3		
Gastrotomy for				
Gastritis	5	5		
Gastritis, hemorrhagic	3	3		
Gastric neuroses	2	2		
Gastric linitis	1	1		
Foreign body	1	1		
Total	12	12		
Gastrorrhaphy				
Ulcer, gastric, perforated	3	3		
Duodenorrhaphy				
Ulcer, duodenal, perforated	5	4	1	
Dilatation				
Esophagus	1	1		
Gastroplication				
Gastrectasis	1	1		
Exploratory for				
Pylorospasm	2	2		
Volvulus	1	1		
Esophageal varices	2	2		
Gastric neuroses	2	2		
Cyst, abdominal	1	1		
Hematoma, abdominal	1	1		
Total	9	9		
GRAND TOTAL	582	554	28	4.81

Although the statistics from general hospitals are by no means so good as those obtained by individual surgeons, yet they are bound to improve as the average surgeon becomes more skillful both in selecting his cases for operation, as well as in the actual performance of the operation itself. The figures of some surgeons show an almost vanishing mortality: in the above lists are several large series of consecutive operations without a single death. It must be remembered, moreover, that these figures include not only operations done on stomachs comparatively slightly diseased, but also many operations done on stomachs very extensively diseased—dilated, distorted or contracted—by chronic ulceration; that the resistance of such patients may be expected to be less that that of the class we are now considering, and the operative mortality is constantly lessening as surgeons become more expert and as their experience increases. This progressive improvement is well shown in the figures published by Hartmann (1905) with characteristic courage: His first series of operations, done while he was assistant to Terrier, comprised 21 gastroenterostomies, with 5 deaths, a mortality of 23.7 per cent., a death rate which is not much higher than that of the average general hospital at the present day. Hartmann's second series of operations, embracing those done from the time he ceased to be assistant to Terrier until Jan. 1, 1903, consisted of 34 gastroenterostomies, with 3 deaths, a mortality of 8.8 per cent.; while a later series, running from 1903 to 1905, including 47 gastroenterostomies, with only 3 deaths, showed a mortality of 6.3 per cent. If, then, to these factors of improvement in the surgical treatment itself, we add that important one of resort to surgical treatment before the patient has become a physical wreck, the contrast between the success of surgical and the ultimate failure of medical treatment becomes even more marked. Hartmann gives the following interesting figures, which for the credit of physicians are not, we are happy to say, very recent. In cases of gastric disease seen and treated primarily by himself and his colleagues—surgeons—the mortality of operation was two per cent.; but in a series of cases which had been primarily treated medically, and which were later referred to Hartmann by their physicians, the mortality of operation was 24 per cent. The two series of cases included sixty patients. Kocher wrote: "The majority of practitioners do not sufficiently realize what brilliant results are to be obtained by operative means in chronic affections of the stomach, commonly known as gastric catarrh. Not only can the numerous dangers of ulcerating affections of the stomach, such as hemorrhage, perforation, transition into cancer, be prevented, but the

disease and its results may be so rapidly and certainly cured that the medical treatment of obstinate cases must be put in the background. . . . The pain in the stomach disappears immediately after the operation. This is the invariable rule. . . . . The patient does not require to pay any further attention to the nature of his food. The vomiting disappears. The bowels become regular. Repeated investigation of the gastric contents shows that there is a progressive improvement in the process of digestion; hyperacidity diminishes; if too little acid is present, it becomes increased, a statement which is in accord with Steudel, Carle and Fantino, Kausch, Hartmann, Soupault, and Mintz." Such words as these, from a surgeon such as Kocher, who weighed well what he wrote and knew whereof he spoke, should be instilled into the mind of every medical man who has cases of chronic gastric indigestion under his care.

If the immediate results of surgical treatment are such as have been described, what are the ultimate results? Is this condition of improvement maintained? Are the cures permanent? A decision in figures in this instance is not so easily reached. This is true not only because of the difficulty, which always exists, of tracing hospital patients after operation; but also because it has become apparent within the last few years that only ulcers at or near the pylorus are improved with any constancy by means of gastroenterostomy; other ulcers may be improved at least for a time but usually are not permanently or markedly benefited. Thus it is that in massed statistics, including operations for all varieties of benign diseases of the stomach, the ultimate results do not appear to be quite so excellent as earlier reports indicated. For example, Bourne reports the end results of gastroenterostomy in 68 patients; of the whole number, only 51 per cent. were cured. But of the pyloric and duodenal ulcers 70 per cent. were cured, while of the ulcers in the body of the stomach only 38 per cent. were cured. Now before operations on the stomach were done with any great frequency only the patients suffering with more or less obstruction of the pylorus were subjected to gastroenterostomy; and it is precisely this class of patients who derive most marked relief from this operation. In recent years, however, this operation has been extended also to classes of patients who, though they may be improved by it, certainly do not show such marked and rapid cures as are seen in patients with pyloric stenosis. The result is, that in comprehensive statistics the end-results of operation sometimes seem to be less satisfactory than was the case five or six years ago. The figures of the senior author's patients fortunately exhibit a progressive increase in the proportion of

cases cured. These patients have all been traced for a year or more after operation.

In the following Table we have assembled recent statistics bearing on this point. Unfortunately most surgeons do not classify separately their results in cases of pyloric and those in cases of non-pyloric ulcer.[1]

End Results of Operations for Benign Diseases of the Stomach

Operator	Date	Cases traced	Cured and much impr.	Per cent.
Bamberger	1909	126	81	64.2
Bidwell	1911	50	35	62.5
Bourne	1913	68	35	51.4
Calderara	1911	99	64	64.0
Deaver	1900–1904	64	49	76.5
Deaver	1905–1907	31	26	83.8
Deaver	1908–1919	224	197	87.9
v. Eiselsberg	1914	248	176	71.0
Finney	1915	152	134	88.1
Galpern	1911	48	27	56.2
Graham (Mayo Clinic)	1911	162	130	80.2
Gray	1909	53	49	92.4
Kocher	1912	43	33	76.7
Krabbel and Geinitz	1914	99	80	80.8
Kümmel	1912	50	47	94.
May	1910	65	32	49.2
Scrimger, Archibald and Pirie	1914	42	17	40.4
Sherren	1920	622	538	86.5
Short	1911	35	20	57.0
Turner	1912	155	117	75.4
Webb	1915	41	...	89.0 (male) 48.4 (females)

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Calderara: Riv. Ven. di Sc. Med., 1911, liv, 452.
Deaver: Records of the Lankenau Hospital, Philadelphia.
v. Eiselsberg: Surg. Gyn. and Obst., 1914, xix, 555.
Finney: Surg., Gyn & Obst., 1914, xviii, 273; Am. J. Med. Sc., 1915, ii, 469.
Galpern: Arch. f. klin. Chir.,1910–11, xciv, 870.
Graham: Boston M. and S. J., 1914, clxx, 221.
Gray: Lancet, 1913, i, 718.

[1] Eusterman, according to C. H. Mayo (1914), has studied the end results from the Mayo Clinic, and finds 92 per cent. of the cases of gastric ulcer with obstruction were improved and 74 per cent. of gastric ulcers without obstruction were cured or improved by operation.

Kocher: Deutsch. Zeit. f. Chir., 1912, cxvi, 183.
Krabbel and Geinitz: Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1914, xxvii, 859.
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Sherren: Surg., Gyn and Obst., 1914, xix, 564.
Short: Bristol M. J., 1911, xxix, 220
Turner: Northumberland and Durham M. J., 1912, xx, 60.
Webb: Arch. Middlesex Hospital, London, 1914–15, xxxii, Clin. S. xiv, 10.

Denéchau (1907) reported his study of the end results of gastro-enterostomy for benign disease, in 104 patients, operated on by different surgeons. He found "satisfactory" results in 54 per cent., moderately good results in 38 per cent., and bad results (no improvement) in only 7 per cent. of these patients.

Paterson traced (1906) the subsequent history of 116 patients who had been operated on by gastro-enterostomy at periods varying from two to nineteen years. He concluded that over 85 per cent. are completely relieved, and 7 per cent. almost completely relieved, thus giving less than 7 per cent. of cases in which the results were wholly unsatisfactory. He found, moreover, if from this series were excluded those cases in which the anastomotic opening was small or in which some mechanical appliance was used to effect the anastomosis, that the proportion in which the result had been completely satisfactory was 92 per cent. This is certainly a favorable showing compared to relapses in 50 per cent. or more of patients treated by medical means.

C. H. Mayo (1920), basing his remarks on experience with 2431 patients, of whom all but 108 were traced, states that after operation for gastric ulcer, the average death rate during the succeeding four years is slightly more than three times normal; while after operation for duodenal ulcer it is slightly less than normal.

We do not wish, however, to be understood as urging surgical intervention in every case of gastric ulcer. As has already been stated, medical treatment always should be tried first, and only when methodical and energetic medical treatment has failed to cure the patient, after it has been persisted in for a reasonable time, or when several temporary cures have resulted in ultimate relapses, only then, we repeat, is surgical treatment to be considered in patients with actively ulcerating lesions. In ulcers such as these, it is mainly on account of the complication of hemorrhage that the surgeon's advice is sought. Perforation is universally acknowledged to call for surgical intervention at the earliest possible moment. But in regard to hemorrhage there is still dispute.

Prognosis in Cases of Gastric Hemorrhage.—As was pointed out in connection with the symptomatology of gastric ulcer, there are several distinct varieties or types in which bleeding from gastric ulcers occurs. The hemorrhage may be profuse and overwhelming; in such cases it has usually been found to be due to very small acute ulcerations. This form of hemorrhage is not readily amenable to surgical treatment. and usually subsides by medical measures, such as astringents, absolute rest, and the local application of ice. Direct transfusion of blood is a most valuable recent addition to our treatment of these cases. It should not be postponed too long (see Case History, p. 81). Hemorrhages which are frequent and slight in amount gradually sap the vitality, and, because often undetected, cause a profound anemia. Occult blood in the feces may be the only evidence of this recurring bleeding. For the relief of bleeding such as this, medical measures are of no avail. The bleeding persists, the patient loses ground, becomes wasted, anemic, thirsty, feverish. The descent may be easy, but it is so merely because it is gradual. It is none the less progressive and sure. What is lost is not regained, and the attending physician will realize, perhaps too late, that the decline into which his patient has fallen is not only irremediable by medical measures, but may even have reached the stage where the shock of an operation will kill. Surgery—successful surgery—cannot be done on patients who have no blood; and it is the physician's duty to learn before it is too late that only surgery can afford relief. Direct transfusion of blood even now may bring back such patients from the verge of the grave; but their attending physicians should not allow them to descend so low. It can be said without any hesitancy whatever, that when such patients are operated on in good time they are restored to health and happiness with a regularity of success which is one of the greatest triumphs of modern surgery.

The other form of hemorrhage to which patients with open ulcer are liable, occurs more frequently than that just mentioned, and is characterized by the intermittent, and by no means regular, occurrence of hematemesis. The vomitus may at times be only streaked with blood, or there may be an attack of vomiting of nearly pure blood, occurring once in six weeks or two months, or even less often. In pronounced and recurrent hemorrhage the patient fails in health so rapidly that radical measures are as a rule willingly undertaken; but in the less severe cases of hematemesis the strength may be partially regained in the intervals, so that the appearance of health is maintained for some time; and the patient, and the physician as well, is often

deluded into thinking that occasional vomiting of blood, with annoying though rather mild indigestion during the intervals, is less of an evil than would be resort to a surgical operation. Could such persons know the uniformity with which such symptoms are relieved by an operation they would be eager for its adoption.

Case.—Mrs. S. W., aged 37 years, admitted to the German Hospital December 7, 1905. In June, 1905, this patient had been treated in the medical wards of the German Hospital for severe hematemesis, having vomited 2000 cc. of nearly pure blood. She had also blood in her stools. Her hemoglobin was 25 per cent. She did well under medical treatment, and refused operation when she became strong enough in our judgment to undergo one. She returned to her home, and lived in comparative comfort until December, 1905, when, after feeling uncomfortable and ill at ease for a few days, she suddenly vomited 1500 cc. of bright blood. She was at once brought to the German Hospital, and soon after admission, on December 7th, she vomited 2000 cc. of blood. She was nearly exsanguinated, but after receiving 3500 cc. of saline solution intravenously, appeared somewhat improved. Her hemoglobin was 43 per cent. on December 8th. By the 13th it had fallen to 31 per cent., in spite of energetic medical treatment. On December 16th, a posterior gastro-jejunostomy with no loop was done, and the patient stood the operation well. Two days later, however, on December 18th, she died of exhaustion, with no further bleeding from the stomach.

Evidently in this patient the operation was done too late to be of any service; she might as well have died without an operation. For as has already been remarked successful surgery cannot be done on patients who have no blood, and the case of this patient is a striking example of the truth of this statement, and teaches a useful lesson.

The **prognosis in cases of gastric perforation** depends almost entirely on the promptness and efficiency with which operative treatment is undertaken. The results of operation for this condition will be fully discussed under the subject of treatment (page 120), and it remains at the present time only to say a few words in reference to certain other circumstances which are held to bear some relation to the prognosis. C. Brunner (1903) laid stress on the influence exerted on the prognosis by the amount of hydrochloric acid in the stomach at the moment of perforation. The prognosis, he found, is most favorable when the hydrochloric acid is most abundant, that is from one to one and a half hours after meals; since under these circumstances the gastric contents are less septic than immediately (one-quarter to one-half an hour) after meals, at which period of digestion the amount of hydrochloric acid in the stomach is extremely slight, and peritonitis therefore more likely. Of course perforation of an empty and nearly sterile stomach is so much the less dangerous. The very great fatality which attends perforation in cases of gastric cancer is probably owing to the septic nature of the stomach contents due to the absence of hydrochloric acid.

Treatment

Having, in the previous paragraphs, attempted to show which cases of gastric ulcer should, and which should not be subjected to operation, it now becomes our duty to attempt to reach a decision as to what special form of operation is to be employed for the relief of the conditions already described. The technical details of the operations discussed will be found described in Chapter XIV.

Under the general term **gastro-enterostomy** (an anastomosis between stomach and bowel) may be included the operation of *pyloroplasty* and Finney's modification of the same, known sometimes by the cumbrous name of *gastro-pyloro-duodenostomy*, as well as Kocher's lateral *gastro-duodenostomy*, and the many and various modifications of *gastro-jejunostomy*. Of these procedures there are only two—Finney's pyloroplasty, and gastro-jejunostomy—which in our opinion merit serious consideration. And inasmuch as the same operations are employed in other affections of the stomach, which will be described in subsequent chapters, it will be most convenient to dicuss at some length, in the present place, the various inherent advantages and disadvantages of these operations. *Pyloroplasty* as modified by Finney (1902) is really an extension of the Heineke-Mikulicz operation. As Mikulicz stated before the Philadelphia Academy of Surgery, in 1903, the usual illustrations of pyloroplasty given in the text-books do not accurately represent the operation, as the incision should be made much longer, so as to extend both into the stomach and the duodenum, and on the lower rather than the anterior wall of the pylorus, thus approaching very closely to the more elaborate operation employed by Finney. The theoretical advantages of such an operation are many. By this method the normal gastro-intestinal channel is not altered, the ingested food passing at once from the stomach into the duodenum, as in the natural state; the operation usually is not difficult to perform, and the subsequent development of regurgitant vomiting is very unusual; it may also be admitted that the enlargement of the pyloric orifice of the stomach will secure to the ulcerated area all the benefits which are now believed to result after lateral gastro-jejunostomy from the admixture with the gastric secretion of bile and pancreatic juice. In the hands of most surgeons, however, the death rate of Finney's pyloroplasty has been higher than has been that of gastro-jejunostomy; but in Finney's own hands the mortality has been lower: in 1914 he reported 100 pyloroplasties with 5 deaths; and in 1915 100 gastro-jejunostomies with 7 deaths. The ultimate results in Finneys' own

cases have also been more satisfactory after pyloroplasty than after gastro-jejunostomy: 78 patients traced one year after pyloroplasty, showed 73 or 93.6 per cent. satisfactory results; while among 74 patients traced more than one year after gastro-jejunostomy there were 61 or 84 per cent. satisfactory results (Finney and Friedenwald). Other surgeons have been very slow to adopt Finney's operation, being contented apparently with the results of gastro-jejunostomy. Mayo in 1905 reported that among 58 patients treated by Finney's method there were only two (3.4 per cent.) secondary operations required, these being for chronic regurgitation of bile into the stomach, through too large an opening; a record which shows that not only were his cases carefully selected, but that the technical details of the operation were carried out with scrupulous care.[1] It is needless to say that it is this very selection of cases which is the most difficult part of surgery. Finney writes: "The only contraindications to the operation are inability to mobilize the duodenum when adhesions are too dense, and thickening and infiltration about the pylorus due to hypertrophic forms of ulceration." The operation, he thinks, "has its greatest indication in the relief of pyloric stenosis due to chronic ulcers, situated at or near the pylorus, and on either side of it, or resulting from the cicatricial contraction following the healing of such ulcers. It is often," he adds, "a useful procedure in cases of hemorrhage due to gastric ulcers on the lesser curvature or to duodenal ulcers which cannot be controlled medically, and which threaten the life of the patient, as well as in the chronic dyspepsias due to ulcers which have not been relieved by medical treatment." He claims special advantages for the opportunity which this operation affords to excise accessible ulcers after direct inspection of the part affected.

In our opinion Finney's method of pyloroplasty should be employed only in patients where perigastric adhesions are absent and where the plyorus is not involved in cicatricial tissue. It will be seen therefore, that there are very few cases indeed in which we deem this operation advisable. Adhesions are Nature's safeguard, and should be treated with respect. In not a few cases the adhesions are on guard over a threatening perforation or over one which had perforated before, subacutely or chronically. In such cases injudicious destruction of adhesions may open up a perforation into the stomach which it may be impossible to close by suture, and in any event this procedure will subject the patient to the risk of septic peritonitis from the unexpect-

[1] In the first edition of this book we quoted also the results of Mr. Rutherford Morison's operations; but we are informed by Mr. G. Gray Turner that these operations were Heineke-Mikulicz pyloroplasties, and not instances of Finney's operation.

ed, and at times undiscovered, extravasation of gastric contents. The safer course is to perform gastro-jejunostomy in a healthy portion of the stomach wall, and leave Nature's barriers undisturbed. The more marked the pyloric stenosis, the more certain are the benefits to be derived from gastro-jejunostomy; and where the pylorus is much obstructed it is involved in cicatricial tissue, and is an extremely unsuitable site for direct incision and suture. Stitches do not hold well in scar tissue, and scar tissue does not lend itself so readily to an anastomotic operation as does normal serous tissue, both because of rigidity and of the lack of blood supply. Yet in cases in which the pylorus is an obstructive factor without being ulcerated or the seat of cicatricial tissue, Finney's operation may prove of value.

Gastro-jejunostomy, on the other hand, has been proved by clinical experience, the true criterion of success, to fulfill most admirably the indications in the surgical treatment of gastric ulcer. The death-rate immediately due to this operation is extremely low, varying from three to less than one per cent. in the hands of experienced operators, and averaging probably not much over ten per cent. in collective statistics. But it should be well recognized that its ultimate results are most satisfactory in cases of pyloric ulcer, especially if there is pyloric obstruction. It is not the best operation for all ulcers in other parts of the stomach (Fig. 36), but it is undoubtedly, even for these, the least dangerous and the most generally applicable operation. If the anastomosis is made in the pyloric portion, not in the body of the stomach, the anastomotic opening will functionate even where the pylorus is patulous (Hartmann, 1914); and even if the gastric contents do not leave the stomach by the new opening, but still are discharged by the pylorus, the gastro-jejunostomy aids in healing the ulcer by permitting admixture of the bile and pancreatic juices with the stomach contents, thus diminishing hyperacidity.

There remains for consideration the treatment of gastric ulcer by **excision.** Originally advocated by Rydygier, it is a method which quickly fell into disrepute, owing to its enormous mortality. But it was again revived by a number of surgeons (Maydl, Jedlicka, Ali Krogius, Rodman, Bréchot, and others), chiefly on the ground that it acted as a preventative of carcinomatous degeneration, but also because it was claimed that excision of the ulcer, or even, if necessary of the whole ulcer bearing area (Rodman, 1900), obviated the occurrence of subsequent hemorrhage or perforation, calamities which are not entirely unknown after subsidence of symptoms procured by gastroenterostomy. Témoin (1917) speaks of gastro-jejunostomy as a mere makeshift

when gastrectomy proves impossible; and there is much truth in Sherren's statement (1920) that gastro-jejunostomy is of value only in the case of an ulcer that is unadherent to a neighboring viscus. Sherren reports that he now adopts excision in 50 per cent. of his cases; and we have found ourselves being gradually forced into the belief that in

FIG. 36.—Skiagraph Three Years after Gastro-jejunostomy, Showing Ulcer on Lesser Curvature, near Cardia, still Unhealed. Symptoms not Relieved. Small Indurated Area with Slight Crater. Bismuth Leaving Stomach through Stoma. (*Lewis Gregory Cole, in American Journal of Roentgenology, Nov.*, 1915.)

all cases the surgeon's preference should be for excision, and that gastrojejunostomy should be employed only where excision would be particularly difficult or dangerous. But even at the present day the mortality of excision remains higher, being 3 to 10 per cent., instead of

1 to 3 per cent. in experienced hands, as it is in the case of gastro-jejunostomy. The immediate mortality of *partial gastrectomy and excision* for benign disease is shown in the following table.

PARTIAL GASTRECTOMY AND EXCISION FOR BENIGN DISEASE

Operator	Operations	Deaths	Mortality per cent.
Deaver (1909–1920)	185[1]	8	4.32
Mayo Clinic, St. Mary's Hospital (1915–1919)	606	18	2.95
Moynihan (1909–1920)	122	3	2.46
Rodman (1910–1915) (collected cases)	205	18	8.7
Sherren (1920)	86	6	6.9
Témoin (1917)	273	8	2.9

[1] The operations by the senior author assembled above under the general heading "partial gastrectomy" may be disassembled into the following subheadings:

Lankenau Hospital (1909–1920)	Operations	Deaths	Mortality per cent.
Partial gastrectomy	38	3	7.8
Pylorectomy	60	3	5.0
Cylindrical gastrectomy	16	1	6.25
Excision	71	1	1.4
	185	8	4.32

Excision of an isolated ulcer is a less severe operation than partial gastrectomy, even when combined with a gastro-jejunostomy as is almost always the case. But as gastric ulcers are often multiple, some may be overlooked, unless a formal excision of the ulcer-bearing area is done (a typical partial gastrectomy); and it may prove impossible to remove all the ulcers without doing a gastrectomy of prohibitory extent. Moreover, even after the excision of the suspected ulcer or ulcers fatal hemorrhage or perforation have occurred from ulcers which were left (Billroth, v. Eiselsberg, Mayo Robson and others). Rodman (1915) collected 171 instances of excision of gastric ulcers, with only 3 deaths, a mortality of 1.75 per cent. The reports of St. Mary's Hospital, Rochester, Minn. (1915–1919 inclusive) tabulate 515 operations of excision of gastric ulcers (including the so-called *cautery excision*, p. 116), with only 10 deaths, a mortality of less than 2 per cent. As already noted, among 71 such operations by the senior author there has been only one death. *Transgastric excision* of ulcers on the posterior wall of the stomach may be adopted in cases where the posterior wall cannot otherwise be made accessible (see p. 364). It has

been commended by v. Eiselsberg, who reported 12 such operations with 4 deaths; and by Kümmel who recorded 18 operations with only 2 deaths. Kümmel traced 9 of his patients and found they were free from symptoms. The operations of this nature by the senior author are included under the heading *excision* in the Table given at p. 113.

The matter of malignant degeneration or carcinomatous implantation in gastric ulcers will be discussed at length in connection with the etiology of carcinoma; but it seems fair to conclude that some microscopical errors may have been made where gastric ulcers have been said to have presented evidences of incipient malignancy, just as clinical errors have been committed in condemning to an early grave patients with large pyloric tumors seemingly characteristic of cancer, which tumors have gradually and quietly melted away after gastro-jejunostomy and other palliative operations. Such cases have been observed by Terrier, Bidwell, Deaver, Wallis, v. Eiselsberg, Robson, Demoulin and Tuffier, Moynihan, Pantzer, Mayo, and many others. Deaver's patient, operated on as a last resort by anterior gastroenterostomy, for a supposedly cancerous mass, was still in excellent health more than six years after the operation. But the fact that carcinoma may develop subsequent to gastro-jejunostomy for supposedly benign disease is a fact that cannot be ignored, and is one of the strongest arguments in favor of excision. Jedlicka pointed out that of the 14 patients whose stomachs were resected by him between 1891 and 1901 for carcinoma, not one was alive in 1904, when he wrote; whereas of the four patients whose stomachs were resected in the same time for supposedly benign disease, which was afterwards however found by the microscope to be malignant, but in an early stage—that of these four patients, the first was well eight years after operation, the second was well four years after operation, the third was still too recent to count; and only *one* died of recurrence, and then only after two circular resections of the stomach.

In reported statistics, however, where no distinction is made between operations for *gastric* and for *duodenal* ulcer, it must be borne in mind how rare is carcinoma of the duodenum, and yet how much more numerous are gastro-jejunostomies for duodenal than for gastric ulcer so that, as Coffey (1920) points out in his own statistics, the subsequent development of one case of carcinoma of the stomach in a series of 165 gastro-jejunostomies implies really that it occurred among approximately 40 such operations for gastric ulcer, the other three-fourths of the operations having been done for duodenal ulcers.

The senior author has seen during the past 20 years five cases in

which carcinoma of the stomach developed after gastro-jejunostomy: in three cases the original operation had been done by himself, at the Lankenau Hospital, and in the two other cases it had been done elsewhere. In two of these patients, mentioned in our first edition (Vol. I., p. 119) death occurred at intervals of four and two years respectively after gastro-jejunostomy by the senior author, and though the cause of death was not determined by autopsy, it was thought to be carcinoma. In the case of all three of the patients seen since the date of our first edition (1909), recurrence of symptoms occurred in less than a year after the primary operation (gastro-jejunostomy), which only in one of the three patients had been done by the senior author. In this case recurrence of symptoms and re-operation took place so soon (about four months) after the first operation, that it is practically certain carcinoma was present at that time but was not recognized. Moreover, in three other cases in which at the first operation portions of the stomach had been removed (partial gastrectomy) for supposedly benign disease, and in which the clinical diagnosis was confirmed at the time by the pathologist, the patients later came back with obvious gastric carcinomata: more sections of the original specimens, made by Dr. Reiman, did reveal an early carcinoma in one of these cases, but serial sections, fairly complete, failed to show any evidence of carcinoma in the original specimens from the two other patients.

Carcinoma Developing Subsequent to Gastro-jejunostomy for Supposedly Benign Disease

Author	Cases of gastro-jejunostomy traced	Developed carcinoma	Per cent.
Bidwell	56	2	3.57
Czerny	53	3	5.66
Deaver (1920)	271	3	1.1
v. Eiselsberg	248	13	5.21
Krabbel and Geinitz	99	4	4.04
Mayo Robson	97	4	4.12
Peck	58	1	1.7[1]

[1] In another patient of Peck's series, carcinoma developed after excision of benign ulcer. Among 135 supposedly simple ulcers excised by Sherren (1920) the microscope found carcinoma in 6.

As Moynihan (1920) points out the mortality from the later development of carcinoma in cases of gastro-jejunostomy for ulcer of the stomach is as high as the mortality of excision would have been if employed as the original operation.

Those gastric ulcers which we think best suited for treatment by excision are those at some distance from the pylorus; for although a number of these patients are often considerably benefited by gastro-jejunostomy, yet it has become perfectly evident that no such gratifying change in their symptoms is produced as when the ulcer is seated at the pylorus. We readily acknowledge that we adopt excision, or even partial gastrectomy, in a very much larger proportion of patients than was the case a few years ago; and are prepared to go so far as to say that in cases of indurated ulcer, no matter where situated, the surgeon's first choice should be for excision of some form: pylorectomy, or partial gastrectomy for pyloric ulcers, wedge-shaped resection or cylindrical gastrectomy for ulcers on the lesser curvature, and transgastric excision for ulcers on the posterior wall, otherwise inaccessible. But though these methods of procedure should be the surgeon's first choice, he should be conservative in his selection of cases, and should attempt excision only in the absence of encumbering adhesions, and in patients not too ill to withstand what often proves to be a somewhat tedious operation.

In many cases where excision would be difficult or impossible, it is relatively easy to destroy the ulcer by the *actual cautery*, according to the method of Balfour (1914): if an ulcer is situated along the lesser curvature, for example, the gastro-hepatic omentum is carefully detached from the ulcerated area, and this area is burned through with the cautery, until the entire indurated area is destroyed. The resulting defect is sutured as if it were a perforation, and these sutures are reinforced by attaching over them the gastro-hepatic omentum. Thirty-seven operations of this kind were done in 1915 at the Mayo Clinic, where the method originated, without a death.

The question of **occlusion of the pylorus** as an aid to gastro-jejunostomy in the cure of gastric ulcers still is unsettled. It is quite well recognized, as has been stated already, that gastro-jejunostomy is most efficient in the cure of ulcers situated at the pylorus and accompanied by pyloric obstruction. Hence, apparently, arose the idea that artificial occlusion of the pylorus would be an aid to gastro-jejunostomy in healing ulcers situated in other parts of the stomach, and unattended by pyloric stenosis. Various methods of occluding the pylorus have been employed, but experience has shown that none of them except formal section and closure of both ends (so-called "exclusion of the pylorus," first employed in 1895 by von Eiselsberg) is permanent in its effects. The simplest method is to infold the anterior wall of the pyloric canal by a series of sutures so as to plicate it longi-

tudinally (Kelling, Mayo, 1900). When adhesions are few a purse string of linen may be passed completely around the pylorus, and drawn tight enough to occlude the channel; if it is not drawn tight enough the channel of course will not be occluded even temporarily, and if it is drawn too tight the ligature will soon ulcerate its way into the lumen of the bowel with restoration of the natural channel. The same uncertainty applies to the aluminum bands suggested by Brewer (1914). Other surgeons have incised all the coats but the mucous, have separated this as a tube without opening the lumen of the pylorus, and have finally ligated this mucous tube and closed over it the muscular and serous coats. Others again have gone one step further in a similar operation, and have ligated this mucous tube in two places, dividing the mucosa between the ligatures, thinking thus to make a permanent occlusion of the pylorus (Biondi, Lewisohn). Still others (Wilms, Hoffman, Strauss, Polya, Bircher) have employed a free transplant of fascia (fascia lata, sheath of rectus, abdominis, round ligament of liver, etc.) to ligate the pylorus, either without incising its walls, or after dissecting the mucous coat as a tube after incising the serous and muscular tunics. But clinical and experimental observations have made it fairly certain that none of these methods, except v. Eiselsberg's, maintains occlusion for more than a few weeks on the average. Many surgeons are contented with this, and contend that this is quite long enough, as all the benefits derived from pyloric occlusion are to be anticipated in the time immediately following operation, and assert that permanent occlusion is not to be desired. In our own mind it remains extremely doubtful whether temporary occlusion of the pylorus is of any value as a primary operation. In cases of "vicious circle" following gastro-jejunostomy, which fortunately are very rarely seen at the present time, we have employed ligation of the pylorus a number of times, and have reason to believe that it was the cause of relief of symptoms. The only theory on which pyloric occlusion can be held to be of value as a primary operation is that which teaches that gastro-enterostomy is of benefit not by admitting to the stomach an excess of alkaline duodenal secretions (the theory which heretofore has had most to support it) but merely by accelerating the evacuation of the stomach, and thus lessening the time during which peptic corrosion of the ulcers can take place. Sippy (1915) holds the latter view; he teaches that as pepsin acts only in an acid medium, and as the acidity of the gastric juice depends very largely on the presence of food in the stomach, the only good gastro-enterostomy can do is to accelerate the evacuation of food from the stomach; so long as the pylor-

us is even partly open, he argues, little or no gastric contents will pass by way of the anastomotic opening, and it is therefore of little value; but if the pylorus is occluded (by stenosis from ulceration, or by ligation, plication, etc.) then the anastomotic opening serves for evacuating the stomach, and accomplishes this in less than the normal time. He overlooks the fact, we believe, that in duodenal ulcer the stomach is often emptied through the pylorus in less than the normal time and yet that the addition of a gastro-jejunostomy promotes healing of the ulcer without further accelerating the speed of evacuation; the only reasonable conclusion it seems to us is that the benefit of gastro-enterostomy is due to the increased alkalinity of the gastric contents secured by admission to the stomach through the anastomotic opening of the alkaline duodenal juices. Moreover, an anastomotic opening properly placed (in the pyloric portion) functionates even in the presence of an open pylorus, as shown by Hartmann (1914). Therefore it remains doubtful whether primary occlusion of the pylorus is of any value.

Sherren (1920), who says he has never done an exclusion of the pylorus, has had eight autopsies from 2 to 9 years after operation (gastro-jejunostomy) for gastric ulcer, and in all cases the ulcer had healed perfectly; moreover in 13 patients he saw the stomach at subsequent operations from 2 weeks to 7 years after the primary operation; and in every case the ulcer had healed.

In stomachs extensively contracted from chronic ulceration, v. Eiselsberg (1897) advocated the palliative operation of **jejunostomy. Duodenostomy,** above the bile papilla, has been urged by Hartmann (1903) as a better operation. Bullitt's patient (1907) was in fair health ten months after operation. R. S. Fowler (1916) reports a case of jejunostomy for extensive callous ulceration involving most of the greater curvature of the stomach, which had brought the patient to death's door; relief was immediate, the tube being worn for six months, and the patient then reporting in normal health, which was maintained at the last report about three years after operation. Patients such as these, in whom no operation of any magnitude can be employed, are often relieved at least temporarily by jejunostomy; and though it is probable that most cases have not the happy terminations noted in most of the published instances, nevertheless, if it is possible to lessen the discomforts of the patients for a few weeks before death, the surgeon need not hesitate to resort to this now classical palliative operation. (See also p. 273.)

In regard to the **treatment of hemorrhage,** probably enough has been

said in the section on prognosis, where it was pointed out that surgical intervention is most successful where operation can be done between attacks of hematemesis; and that operations done with any idea of locating and ligating the bleeding point, in cases of acute hemorrhage, fail in the immense majority of instances to accomplish the desired result. Yet Dieulafoy urged operation in these very cases "at the opportune moment." In the sudden, profuse, and overwhelming bleeding sometimes encountered, and which is generally the first and sometimes the only symptom of the "exulceratio simplex" known by his name (see p. 70), he strongly counseled surgical intervention on the first recurrence of the bleeding. The first patient whom he saw with this variety of hemorrhage died from recurrence of the profuse hematemesis shortly after coming under observation. At autopsy the seat of hemorrhage was found in a small arteriole just beneath the muscularis mucosæ, which has been perforated by an "exulceratio simplex." In his second patient he correctly diagnosed the cause of the hemorrhage, and on its recurrence the next morning induced Cazin to operate in the hope of finding and ligating the bleeding point. The stomach was opened and by everting its mucous lining through the incision like a glove on the hand, and by minutely searching among the mucous folds and rugæ, a suspicious looking area was detected. The manipulation and sponging of this area started the bleeding afresh and the arteriole was then ligated, the patient making a good recovery. Robson and Moynihan in 1904 recorded two similar cases in which several bleeding points were successfully ligated. We should feel extremely loath to undertake an operation in cases such as these, where the chance of discovering the seat of hemorrhage is so exceptionally slight, and where medical treatment offers a probability of cure in a fair proportion of cases.

The alarming mortality which attends operations undertaken for the relief of acute hemorrhage may be seen from the following figures quoted from Lieblein and Hilgenreiner (1905): Hartmann reported a mortality of 63 per cent.; Savariaud, 66 per cent.; Robson (42 cases), 64 per cent.; Quénu, 45 per cent.; Kaupe, 40 per cent. (probably mostly chronic recurring bleeding). Munro (1904) out of a series of eight patients operated on for acute hemorrhage saved only one. Moynihan's mortality among 27 operations was nearly 26 per cent. Tuffier says that with medical treatment the mortality from acute gastric hemorrhage is only 1.7 per cent., so that even if some cases included under medical treatment were so mild as never to have been considered surgical, and even if we accept the highest mortality under

medical treatment, that of 11 per cent., given by Müller, yet the difference in the mortality between medical and surgical treatment is too great for surgical treatment to be preferred in patients with acute hemorrhage, save in the most exceptional cases. Direct transfusion of blood usually will not only promptly check the bleeding, but will in most cases render even an extremely anemic patient a fit subject for operation. Rodman (1915) advocated the use of water at a temperature of 130 degrees through the stomach tube; he said in 20 such cases of gastric hemorrhage this treatment had never failed to check the bleeding.

Nor in recurrent hemorrhage, when the operation is done in the interval, is it desirable to ligate or excise the offending ulcer, unless this is callous and indurated, and then only when it is easily accessible. It usually suffices to infold the gastric walls over the ulcer, catching in the sutures any vessels which seem to lead up to the ulcer; this should of course be supplemented by gastro-jejunostomy.

The **treatment of perforation** of a gastric ulcer, is unquestionably operative. Without operation death will be the natural consequence in 99 per cent. of cases. Suture of a gastric perforation was first done by Mikulicz in 1889, but without success. The first instance of recovery after suture of a gastric perforation was recorded in 1892 by Kriege. From statistics given in the first edition of this work it is evident that in the usual run of cases it was at that time (1909) exceptional for more than half of these patients to be saved by operation. We give below in tabulated form, statistics which have been published since the first edition of this book appeared.

Results of Operations for Gastric and Duodenal Perforations (Collective Statistics)

Operator	Cases	Died	Mortality per cent
Caird (Edinb. Med. Jour., 1914, xiii, 455)			
1896–1903	54	34	63.0
1904–1913	193	71	36.7
Hartmann and Lecène (Annals of Surg., 1914, lx, 227)	202	107	52.9
Peck (Jour. Am. Med. Ass., 1915, lxv, 659)	30	8	26.6
Pètren (1894–1910) (Surg. Gyn. and Obst., 1912, xiv, 544)	135	81	60.0
Scully (Am. J. Med. Sc., 1918, clv, 874)	49	28	57.1
Short (Bristol Med. Chir. J., 1911, xxix, 220)	58	26	44.0
Walker (Bost. M. & S. J., 1915, clxxiii, 452)	78	21	26.9
Wetterstand (1900–1910) (Deutsch. Zeit. f. Chir., 1913, cxxi, 393)	60	33	55.5

It seems from these figures that the death rate of this complication is still needlessly high. If all patients were operated on at the most opportune time, that is, within a few hours of perforation, the results would be much better, as may be seen in the accompanying table, taken from the monograph of Gross and Gross (1904) and from that of F. Brunner (1903). In 237 instances noted by Gross and Gross the time between perforation and operation was recorded; and the results, as well as Brunner's figures may be thus presented:

Duration of perforation	Mortality per cent.	
	Gross and Gross	Brunner
Less than 12 hours	25.00	25
Less than 24 hours	52.72	46
Less than 48 hours	56.06	58
More than 48 hours	73.91	80

If the results of individual surgeons are examined it will be seen that the personal equation has something to do with the results. Thus a surgeon who has seen a number of these patients will not only make his diagnosis more quickly,[1] and will hence operate sooner, but the operation itself will probably be performed with greater skill and dispatch than will one done by the occasional operator. The same will be true of a series of operations done in a well-equipped hospital, by various members of the same staff.[2] The following figures accordingly are not without their interest:

[1] In most metropolitan hospitals the Internes on duty in the Receiving Ward rarely fail to make the correct diagnosis.

[2] Of 57 operations for perforations, done by the Senior Author, 42 were for duodenal and only 15 for gastric perforations. A primary gastro-jejunostomy was done in 44 cases, there being 3 deaths in this series, a mortality of 6.8 per cent.; suture alone was done in 8 cases, with one death (12.5 per cent.); and partial gastrectomy was done in 5 cases, without a death.

RESULTS OF OPERATIONS BY INDIVIDUAL SURGEONS FOR GASTRIC AND DUODENAL PERFORATIONS

Operator	Cases	Died	Mortality per cent.
Ashhurst (Episcopal Hospital, Phila.)	10[1]	3[2]	30.0
Alexander (Episcopal Hospital, Phila)	12	2	16.6
Collinson (Jour. Am. Med. Assoc., 1914, lxiii, 1184)	53	20	37.7
Connors (Amer. J. Surg., 1916, xxx, 173)	42	14	33.3
Deaver (German Hospital, Phila., to Jan 1, 1920)	63[4]	4	6.34
Deaver, H. C. (Episcopal Hospital to June 30. 1920)	16	2	12.5
Farr (Annals of Surg., 1920, ii, 591)	25	3	12.0
Gibson (Surg., Gyn. and Obst., 1916, xxii, 388)	14	1	7.1
Kirk (Choyce's System of Surg., 1912, ii, 356)	11	0	0.0
Mayo Clinic (St. Mary's Hospital Reports, Rochester, Minn., 1911–1915.)	27	5	18.5
Moynihan (Bost. Med. J., 1910, I, 241.)	25	5[3]	20.0
Miller (Trans. Phila. Acad. Surg., 1915, xvii, 160)	6	2	33.3
Peck (Jour. Am. Med. Assoc., 1915, lxv, 659.)	12	0	0.0
Ross (Trans. Phila. Acad. Surg., 1915, xvii, 151)	6	2	33.3
Rodelius (Beitr. z. klin. Chir., 1914, xcii, 277)	15	8	60.0
Sherren (Choyce's System of Surg., 1912, ii, 356)	28	12[5]	42.8
Struthers (Edinb. Med. J., 1920, xxiv, 748)	90	20	22.2
Sullivan (Jour. Am. Med. Assoc., 1916, ii, 330)	20	1	5.0
Wagner (Deutsch, Zeit. f. Chir., 1912–1913, cxx, 438)	15	6	40.0

[1] One successful operation by Dr. I. M. Boykin.

[2] Two patients nearly moribund at time of operation; one aged 84 years.

[3] Two other patients moribund when first seen, died without operation.

[4] Seven other patients died without operation. Notes of 4 of these cases follow:

Male, 51 yrs., perforation 2 days before admission, admitted moribund, death in 10 hours. (March 10, 1911.)

Male, 57 years, admitted with diffuse peritonitis, thought to be from strangulated right inguinal hernia. Drainage of abdomen through inguinal canal. Death in 2 days. (April 15, 1911.)

Male, 38 years, perforation 3 days before admission, admitted moribund, death in 10 hours. (Oct. 18, 1911.)

Male, 32 years, perforation 14 hours before admission, while on alcoholic debauch; admitted moribund, and died in 5 hours. (June 3, 1916.)

The diagnosis in all these cases was confirmed by post-mortem examination. If all these deaths are added to the figures above, the mortality is 15.7 per cent.

[5] All the fatal cases, as well as six of the recoveries, came to operation more than 24 hours after perforation.

It thus appears that at the present time from sixty to seventy per cent. or more of patients are being saved, and that with prompt operation the mortality may be expected to be as low as ten or even five per cent.

Gross and Gross also analyzed the results in the series of cases collected by them, so as to include operations done within five hour

periods after perforation. Thus they found that of those patients operated on

Within the first five hours	31.03 per cent. died.
Within the second five hours	16.25 per cent. died.
Within the third five hours	42.85 per cent. died.
Within the fourth five hours	54.00 per cent. died.
Within the fifth five hours	57.14 per cent. died.

This seems to show that operations undertaken within the first five hours are less successful than those done during the second period of five hours; but we think that notwithstanding these figures no surgeon should hesitate to open the abdomen at the earliest possible moment after perforation has occurred. To postpone operation is usually to await the development of an irremediable peritonitis; indeed some patients are so profoundly toxic that they do not survive long enough for peritoneal reaction to occur. The apparent contradiction between clinical experience and the figures obtained on analysis could probably in this instance as in others be satisfactorily explained if the cases had been reported in greater detail; when it probably would have been found that the majority of those patients operated on within the first five hours after perforation, suffered from severer lesions than did the others. Shock in these patients is in large measure due to the toxemia due to absorption from the suddenly inundated peritoneal cavity; and the best method of arresting this is to open the abdomen, close the perforation and relieve the intra-abdominal tension by drainage.

Bearing on this subject the observations of F. Brunner (1903) are of interest. He constructed curves to represent graphically the prognosis after operation in cases of gastric perforation. According to this method he finds that the curve of mortality gradually approaches that of recovery up to the eleventh hour after perforation, when it crosses the curve of recovery, and thereafter exceeds it. The curve of recovery before the eleventh hour is parallel with the curve of abdominal rigidity; while the curve of mortality after the eleventh hour is parallel to the curve of abdominal distention. In other words, at the eleventh hour, the chances of recovery are about 50 per cent., being greater before, and growing progressively less after the fateful hour has been passed. The prognosis also is good so long as the abdomen is rigid; but when absorption of peritonitic toxins has caused abdominal distention, the prognosis becomes progressively worse the longer the time that has elapsed since the subsidence of rigidity. All these ob-

servations render the importance of prompt operation so much the more apparent.

Most gastric perforations are on the anterior wall of the stomach and are fairly accessible. Baker (1917) proposed administering by mouth to patients with symptoms of perforation a dose of 3 grains of methylene blue, dissolved in an ounce of water. The stain, escaping from the perforation, aids in its location. We have had no occasion to employ this method. *Excision* of the ulcer is an unnecessary waste of time[1]; by this procedure, moreover, the surgeon not only leaves himself a larger opening to close, but may also add the complication of hemorrhage to that of perforation, since some good sized vessels may be divided. Sero-serous *suture* of the perforation, without even attempting to freshen its edges, is quite sufficient. It is better to use linen than catgut.

But in certain instances it is impossible to close the opening securely by suture, and in some rare cases the perforation will be so situated or its edges will be so friable that sutures of any kind, even insecure sutures, cannot be inserted. Under such circumstances the surgeon should endeavor to close the perforation by suturing a tag of the great omentum over it, a method which appears to have been first employed in 1897 by Braun. Or the gastro-hepatic omentum may be anchored down to the perforation if more convenient. In cases where such devices fail, the surgeon should not despair of curing his patient, but should pack off the perforated area with gauze pads, as is done in similar circumstances in other regions of the abdomen. This is a much safer plan than attempting to suture the perforation to the abdominal wall. F. Brunner (1903) collected 15 cases of perforation of the stomach treated by packing without suture. Of these no less than 12 recovered; whereas suture of the perforation to the abdominal wall is nearly always followed by death. To these cases of gastric perforation treated by packing, recorded by Brunner, may be added Wood's patient (1904) who also recovered, thus giving 16 recoveries and only 3 deaths for this method of treatment, a mortality of only 18.75 per cent. The resulting gastric fistula has closed spontaneously almost without exception.

Villard and Pinatelle (1904) strongly commended packing for ulcers which have perforated among adhesions close to the lesser curvature. After the packs have been placed the greater curvature of the stomach may be sutured to the abdominal wall if there is doubt as to the

[1] Finney, however, strongly commends excision of pyloric ulcers in the longitudinal axis of the canal, with closure of the opening according to his method of pyloroplasty.

efficiency of the packs. These authors collected 9 cases of this character, treated by packing without suture: all three patients in whom the perforation was drained by a tube into the stomach, packed around with gauze, recovered from the operation; while of the remaining six patients in whom gauze packs alone were used, four recovered and two died. The two fatal cases were in patients operated on respectively 37 and 60 hours after perforation had occurred; the first survived the operation by six and the second by ten days, showing that they possessed an unusually good chance of ultimate recovery.

The abdomen should invariably be *drained.* The surgeon should studiously avoid the example of Young (1905), who, because he thought it impossible to drain well, did not drain at all. His unfortunate patient did well for four weeks in spite of the fact that he had two perforations in his stomach; but finally succumbed in the fifth week to exhaustion due to a large subphrenic abscess.

If the closure of the perforation is secure, the surrounding tissues not being friable, and the sutures holding well, the epigastric incision may be closed without drainage; but in cases where there is the least possible doubt as to the sutures holding it is indispensable to leave a cigarette drain in the upper wound. *In all cases*, whether the epigastric incision is drained or not, a second incision should be made in the suprapubic region, and the pelvis should be drained by a glass tube as in other cases of diffuse peritonitis. This is imperative, because, although in most cases in which operation is done within 12 hours of perforation the peritoneal exudate is sterile, it is not always so. One of us (Ashhurst) has found the colon bacillus in mixed culture in the pelvic exudate within less than 5 hours after perforation of a duodenal ulcer, though clinically there was nothing to distinguish this case from others in which the exudate was sterile.

No *irrigation* of the peritoneal cavity should be employed. We believe this statement holds good even for late cases, or for those where food particles may be recognized in the exudate. The chances of recovery will be greater in these late cases if the surgeon contents himself with sponging out with moist gauze such food particles as are readily accessible.

Some patients are seen so late after perforation occurs that it seems evident that immediate operation will only hasten the *exitus lethalis.* If these patients had been treated from the time of occurrence of the first symptoms of perforation according to the strictest rules of the so-called "Ochsner treatment" it is probable, as already mentioned, that 1 per cent. of them might recover without operation. Perhaps

as many as 5 per cent. more might have been able to localize the infection, and would eventually come to operation for drainage of a subphrenic or perigastric abscess. As they have come into our hands heretofore, they have been moribund, and we have not disturbed their dying moments by resort to what in these circumstances amounts to a cruel and a useless operation. Seven such patients (4 during the last 10 years), who have been under observation for a few hours before death, have perished without the benefits which modern medical science affords for the prevention and cure of peritonitis. These deaths are not chargeable to surgery nor to the surgeon, but to the medical men into whose hands these miserable patients committed their mortal bodies.

When one perforation has been found and closed, should a *second perforation* be looked for? Undoubtedly it should; but if not readily found further time should not be wasted in a search which will prove futile in four out of five cases at least, especially since the time so consumed may be more profitably spent, we believe, in the performance of gastro-jejunostomy.

Gastro-jejunostomy as a primary operation in a patient with gastric perforation appears to have been first employed by Braun in 1897 when he unexpectedly found a perforation in a patient upon whom he was preparing to do a gastro-enterostomy for pyloric stenosis. There are two reasons for performing gastro-jejunostomy as a primary operation: first to promote healing of the perforated area, and second to prevent recurrence of symptoms or a subsequent perforation. Especially important is a primary gastro-jejunostomy if the perforation is close to the pylorus, for its closure will then be very likely to cause obstruction, if not immediately, at any rate when cicatrization has been complete.

There are two important questions to be answered in this connection. First, does the adoption of gastro-jejunostomy affect the immediate mortality of the operation for gastric or duodenal perforation? Second, does its adoption affect the end-results?

When the first edition of this work was published, we could find only 22 instances recorded in which gastro-jejunostomy had been employed as a primary operation. From the following Table it is very evident that it is now accepted by many surgeons as the proper procedure, at least in selected cases; and that its adoption, taking the case reports in general, does not add to the primary mortality. In regard to the end-results less certainty exists, as the reports do not include so many cases; moreover, the end-results of operations without primary gastro-jejunostomy are not definitely known. Twenty of the senior author's patients

Operations for Gastric or Duodenal Perforation with Primary Gastro-jejunostomy. Immediate Mortality

Operator	Operations	Immediate mortality per cent.
Ashhurst (Episcopal Hospital)	4	0
Caird (Edinb. Med. J., 1914, xiii, 455) (Collective statistics)	117	28.20
Collinson (Jour. Am. Med. Assoc., 1914, lxiii, 1184)	27	30.0
Deaver (Lankenau Hospital)	44	6.8
Galpern (Arch. f. klin. Chir., 1910, xciv, 870)	7	14.0
Hartmann and Lecène (Ann. Surg., 1914, lx, 227)	4	0
Hess (St. Petersb. med. Woch., 1910, xxv, 600)	5	80.0
Kroiss (Beitr. z. klin. Chir., 1910, lxvii, 509)	65	21.5
Kümmell (Deutsch. med. Woch., 1912, xxxvii, 395)	5	20.0
Moynihan (Duod. Ulcer, Phila., 1912)	4	25.0
Peck (Jour. Am. Med. Assoc., 1915, lxv, 659)	12	0
Struthers (Edinb. M. J., 1912, ix, 505)	25	25.0
Sullivan (Jour. Am. Med. Assoc., 1916, ii, 330)	10	0

have been traced for more than a year, and all reported themselves free from symptoms. Among Caird's collected cases of operation for gastric perforation, in only 29 of which primary gastro-jejunostomy was employed, the end-results were ascertained in 85 patients, as may be seen below:

	Recovered from operations	Cases traced	No symptoms	Per cent.
1899–1903	17	7	7	100.0
1904–1908	58	36	27	75.0
1909–1913	64	42	42	100.0
	139	85	76	89.4

Caird found, moreover, that among 40 patients treated by suture alone, no less than 19, or 47.5 per cent., were later reported to be suffering from dyspepsia; while of 52 treated by primary gastro-jejunostomy, only 5, or less than 10 per cent., suffered from digestive troubles. This would indicate, he argues, that primary gastro-jejunostomy presents the advantage of warding off dyspepsia. The records, however, showed no evidence, he says, that in any of the fatal cases was death due to the omission of gastro-jejunostomy or that the omission seriously

interfered with recovery. English, as long ago as 1903, traced 17 out of 24 patients who recovered after suture of a gastric perforation, no gastro-jejunostomy having been done in any case: of these 17 patients, 13 had no further gastric symptoms and 4 were dyspeptic, presenting symptoms not of acute ulcer but of a cicatrix and adhesions.

Most surgeons are agreed that in cases where closure of the perforation produces stenosis of the pylorus it is advisable to adopt gastro-jejunostomy as a primary operation. In Caird's duodenal series (88 cases) primary gastro-jejunostomy was obligatory in 39, and was chosen in 49 other cases because of the good general condition of the patients after closure of the perforation. The latter series of cases comprised only 3 deaths, a mortality rate of only a little over 6 per cent.

Struthers states that it was from his cases in which gastro-jejunostomy was obligatory that he first learned how beneficial the operation appears to be in promoting the patient's comfort during recovery. He traced 14 out of 17 patients, and found none of them suffered from serious digestive disturbance, up to four years and a half after operation; but of 3 cases in which gastro-jejunostomy was not done, two patients were well for a time, but later developed symptoms of ulcer, while the third alone remained free from symptoms, which was his condition before perforation had taken place. Sullivan (1916) traced 5 out of 10 patients who had had a primary gastro-jejunostomy, and found all of them free from symptoms; but adds that he did not receive a similar encouraging report from three other patients in whom simple closure of the perforation was done. Peck (1915) concluded that his study of the end-results in a series of cases was not of much value in finding an answer to the question of whether or not primary gastro-jejunostomy should be employed. He adds "It would seem fair to assume, however, that primary gastro-enterostomy in properly selected cases does not greatly increase the immediate mortality, and that it should increase the prospects of ultimate cure." None of the cases in his series treated by simple suture, however, required secondary operations for pyloric stenosis. Collinson (1914) traced 9 patients, gastro-jejunostomy not having been done in any: 4 patients, or 44 per cent. were found to be free from symptoms, 4 had had a secondary gastro-jejunostomy and were then relieved of their indigestion, and 1 still had ulcer symptoms. Of the 4 patients who submitted to secondary gastro-jejunostomy for persistent indigestion, only 1 was found to have true pyloric stenosis, while the 3 others merely had peripyloric adhesions. Collinson also traced 11 patients in whom gastro-jejunostomy had been done as a primary operation:

of these 9, or nearly 82 per cent. were free from symptoms; 1 had died since of carcinoma of the esophagus, and 1 had been operated on recently for gastrojejunal ulcer. Collinson extended his investigations beyond his own patients, and traced also 17 patients of other surgeons, in whose cases primary gastro-jejunostomy had not been employed: 13 were free from symptoms, 4 had recurrence of symptoms. Of 16 cases with primary gastro-jejunostomy 10 were free from symptoms and 6 still had some indigestion. Combining Collinson's personal figures, with those he collected, we have notes of the end results of

26 patients *without* gastro-jejunostomy of whom
17 or 65 per cent. are free from symptoms
9 or 35 per cent. have recurrence of symptoms
27 patients *with* gastro-jejunostomy of whom
19 or 70 per cent. are free from symptoms
7 or 26 per cent. have recurrence of symptoms
1 is dead of another cause.

Collinson's conclusion as to the adoption of primary gastro-jejunostomy is "When in doubt, *Don't.*"

Gibson (1916) traced 7 patients for a sufficient length of time after operation to ascertain end-results: none of these had a primary gastro-jejunostomy, and only one suffered after operation from any gastric symptoms, and in this case they were really negligible.

Other surgeons, however, have not been so fortunate. Paterson (1906) stated that among the cases of gastric perforation which he collected, no less than 13 deaths out of a total of 58 could almost certainly have been prevented if a primary gastro-enterostomy had been done; indeed Paterson goes further than we should be inclined to do, and claims that even purulent peritonitis is no contra-indication to gastro-jejunostomy. In two of Caird's cases, this operation had to be done three days after suture of the perforation, on account of pyloric stenosis; Allingham and Thorpe had to resort to it one month later to accelerate their patient's convalescence; Scudder resorted to it five weeks after, and Gibbon 18 months after suture of the gastric perforation. Mayo and Moynihan have had a similar experience. One of us (Ashhurst) was very thankful that he had employed it as a primary operation in a patient in whom leakage of bile from the perforation occurred along the drainage tract, on the second, third, and fourth days after operation; the gastrojejunal anastomosis permitted prompt and spontaneous closure of the duodenal fistula, with recovery of the patient.

It continues to be our opinion, that in any case where it is not specifically and positively contra-indicated, gastro-jejunostomy should be employed as a primary operation in patients with gastric perforation.

Exploratory Laparotomy.—Operations undertaken in patients suspected of having suffered perforation of a gastric ulcer, must in the nature of things at times be merely explorations. A positive diagnosis is not always possible, and it is usually more to the patient's interest for the surgeon with proper facilities at his disposal, to explore the abdomen, than for an operation to be postponed until the advent of unmistakable peritonitis renders the diagnosis certain. Gross and Cross in their extensive review of the literature (1904) found records of only two patients who died after being subjected to a *laparotomie blanche*, as it has been called—that is to say a laparotomy in which no lesions were found to account for the symptoms. Nor could these two solitary deaths be attributed to the exploratory operation, since death in one was due to persistent hematemesis, for which no cause could be found, and in the other was caused by the rupture of an aortic aneurism. *Laparotomie blanche* has been reported by Kirk (3 cases), by English (3 cases,) and by Körte (2 cases). English also mentions 4 other patients in whom operations were undertaken for gastric perforation, but in whom the symptoms were found to have another cause.

Esophageal perforations into the peritoneal cavity have been recorded by Körte, as well as by Mesnard and Feroualle. Both patients died.

CHAPTER V

BENIGN DISEASES OF THE STOMACH AND DUODENUM
(Continued)

Pyloric Obstruction

Under the general heading of pyloric obstruction it is convenient to group three distinct affections. These are **Infantile Pyloric Stenosis, Pylorospasm,** and **Gastric Dilatation.** Although pylorospasm may possibly be considered merely a symptom, and gastric dilatation a complication or a consequence of gastric ulcer, yet each of them seems of sufficient importance to render advisable its inclusion within the present section.

Infantile Stenosis of the Pylorus

Congenital Hypertrophy of the Pylorus, or Hyperemesis Lactantium, as it has been variously called, is not yet a distinct pathological entity. As early as 1788, Beardsley of New Haven is said, on the authority of Osler, to have recognized this disease clinically, and to have described his findings at autopsy, under the name of scirrhus of the pylorus. Hirschsprung in 1888 reported a case. According to Weber (1910), surgical treatment was first adopted in 1893, by Cordua, who did jejunostomy, but with a fatal result. In 1896 Schwyzer suggested Loreta's operation; and in 1897 Stern operated by gastro-enterostomy. The first successful operation, also by gastro-enterostomy, was performed in 1898 by Löbker. Further references to the literature may be found in the articles of Neurath (1899), Trantenroth (1902), Cautley and Dent (1906), and Wachenheim (1905).

Because there is not yet agreement as to the pathological changes producing the symptomatology, some authors, notably Meinhard Schmidt, have preferred to retain the original symptomatic name Hyperemesis Lactantium. But the trend of modern opinion is toward the adoption of the term Infantile Stenosis, which while not asserting that the condition is a congenital deformity, as some have maintained, nor committing the writer to any clearly defined pathology, nevertheless expresses with sufficient accuracy the changes usually found at operation or autopsy.

Causes.—Three theories have been recognized as to the causation of the symptoms about to be described. These may be briefly denominated the *congenital abnormality theory*, the *hypertrophy theory*, and the *theory of simple spasm*. The first asserts that the pyloric stenosis is a congenital abnormality quite as truly as hare-lip, webbed fingers, or imperforation of the anus. Cautley and Dent state that the pyloric thickening is primary, and due simply to a redundance of tissue, placed there by nature as the result of over-exertion in forming the ordinary sphincter. In support of this theory, Neurath asserted that a family predisposition might exist, quoting Henschel who observed three, and Ashby who observed four cases in the same family. Moreover, in one of Ashby's cases there was also atresia ani, a fact which Neurath thinks lends support to the congenital abnormality theory, not to that of spasm nor to that of hypertrophy. Actual atresia of the pylorus or duodenum has in a few instances been found at autopsy (Cleemann, Eastes, Goodhart, Habheggar, Lesshaft, etc.). Should it by any possibility be recognized during life, it would of course be susceptible of operative relief, even if with very small chance of success. Maylard has called attention to congenital narrowness of the pylorus, not caused by hyperplasia of the pyloric sphincter, as a cause of indigestion in young adults; Mayo Robson has also seen it; and it is of course possible that some cases of hyperemesis lactantium may be due to a similar condition. But that the symptoms of this malady are very rarely manifested before the baby is a week old at least, and that in several instances children no longer infants have developed the disease (Sonnenburg's patient was six years of age), are facts which militate strongly against the assumption that the affection is due solely to any deformity existing before birth. At almost every autopsy and operation at which the pylorus in these patients has been brought to view, the actual condition has been found to be one of increase in the muscular tissue, particularly the circular muscle fibres surrounding the pyloric opening of the stomach, with edema. And what lends further support to the theory that it is an hypertrophic, not a neoplastic, overgrowth, is the fact that on the gastric side the thickening is not sharply limited, but extends for some distance into the pyloric portion of the stomach, as an hypertrophic overgrowth might be expected to do, since the pyloric antrum would naturally be involved in such change; whereas on the duodenal side the thickening ceases suddenly, and within a very short space after the pylorus is passed the duodenum has been found to present its normal characteristics.

To induce this hypertrophy alleged to be the pathological change,

it may be assumed that there is or that there has been a small erosion or fissure in the pyloric region of the stomach, and that there has also been hyperacidity of the gastric juice. That these factors, so well known as causes of pylorospasm in the adult, should in the infant be provocative of a like change, appears in no way unreasonable; and if it be objected that sufficient time does not elapse between birth and the occurrence of the hypertrophy for it to be explained on these grounds, it may with perfect justice be replied that infantile tissues cannot always be judged by standards derived from adult life. John Thomson, the well known pediatrist, has supported the theory of hypertrophy due to incoördination of the muscle during fetal life, and Jedlicka also maintains that prolonged spasm may induce hypertrophy. Meinhard Schmidt compares the condition to that of vaginismus, tenesmus ani, and blepharospasm which are frequently caused by fissure or ulcer. Yet no hypertrophy occurs in such cases, and Rolleston reminds us that no hypertrophy of the pylorus is found in Reichmann's disease, gastro-succorhea, which is usually accompanied by pylorospasm. But as has been said before, it is not always safe to argue from adult to infantile conditions. Pfaundler, while not denying the presence in some of these patients of increase of muscular tissue, considers the condition in most instances merely one of spasticity. Heubner, from a study of 41 cases encountered among 10,000 children, concludes that the affection is due to pylorospasm causing hypertrophy. And Pfaundler now recognizes the existence of two forms—hypertrophic and spasmodic—the latter being intermittent in character. Our own tendency is to hold that the unquestionable increase in muscular tissue is in the nature of an hypertrophy, and is brought about by persistent spasm due to irritation from one cause or another. Similar changes might be expected to be encountered occasionally in other portions of the gastro-intestinal tract; and as a matter of fact one of us (Ashhurst) has recorded (1917)

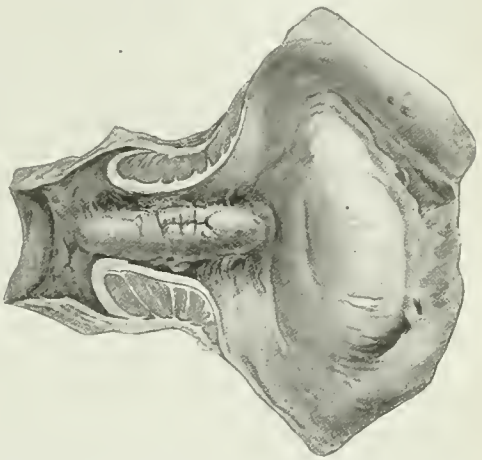

FIG. 37.—Infantile Stenosis of the Pylorus. Natural Size. Note the Thickened Sphincter and the Fold of Mucous Membrane Occluding the Orifice. (*After Cautley and Dent.*)

under the name *ileo-cecal infantile stenosis*, precisely an analogous condition.

The redundance of mucous membrane, to be presently alluded to, may act itself so as to provoke spasm, much as a polypus does in the same and in similar situations. It should, perhaps, be noted that Wernstedt (1907) tried to explain the presence of this mucous fold by studies in comparative anatomy. The existence of primary stenosis of moderate degree, from a congenital abnormality of the pyloric musculature, is held by Downes to be quite sufficient to explain the subsequent development of the pyloric tumor through the action of spasm inducing hypertrophy and edema.

It is to be hoped that further study, both clinical and microscopical, will elucidate these questions, and place the pathology of this serious condition on a firm basis. Until then we must be content to theorize as to the causes, and proceed as best we may, empirically, to adopt uncertain treatment for fairly characteristic symptoms.

The usual appearance of the parts involved is very well shown in Figure 37, copied from Cautley and Dent's article. At first glance the resemblance to an enlarged prostate with the bladder attached, is quite striking; and this becomes greater when on more careful examination we see a fold of mucous membrane which corresponds very closely in appearance to the uvula vesicæ. This mucous fold is a characteristic feature of infantile pyloric stenosis, and often renders nearly complete the obliteration of the passage from the stomach to the duodenum, even when the mere muscular mass would with ease permit the passage of a probe through the pylorus. Meinhard Schmidt (1901) estimated the calibre of the normal pylorus at birth as admitting a No. 19 French sound, and as increasing one number of the French scale (or one-third of a millimetre in diameter) for each month of life; so that a diameter of nine millimetres, equal to No. 27 of the French scale, would be normal for a child of eight or nine months of age, and at twelve months of age the normal pylorus should admit a No. 32 F. sound. According to Fisk, Still has stated that at six months of age the normal pyloric wall is about 2.5 mm. thick; while in hypertrophic stenosis it has been found to vary from 3.5 to 5.7 mm. in thickness. Strauss (1918) has observed that invariably the tumor is larger the older the baby, being small in infants 3 to 4 weeks old and large in those of 7 to 12 weeks.

In only a few instances have there been any microscopical evidences, even slight, of acute inflammation; so that with our present knowledge we are limited to the theories already mentioned of neoplastic and

hypertrophic overgrowth, the latter seeming the more reasonable of the two.

Symptoms.—The symptoms of this affection do not differ materially from those of the same condition in adults. The chief subjective signs are vomiting and constipation, and the chief objective signs are a pyloric tumor and visible gastric peristalsis. The vomiting usually does not begin until the baby is about a week or ten days old, though in rare instances it has been noted from birth, or has not appeared for a month or more. In the earliest stages liquids may be rejected almost as soon as they are swallowed. The vomiting is nearly invariably cumulative as well as projectile, when the disease has lasted more than a few weeks; that is to say, three or four feedings will be retained, and then after the last feeding the whole of the gastric contents will be rejected at once. Hyperacidity is usually present. Bile is conspicuous by its absence from the vomitus. The lack of absorption accounts for the constant hunger, the persistent constipation, and the progressive emaciation. These babies should be weighed at regular intervals. There is no other method which so surely shows the loss of flesh. As emaciation proceeds and the vomiting becomes more marked, as it usually does, there is as a rule very little difficulty in detecting a pyloric tumor; indeed this is frequently visible to the most casual glance, projecting from the sunken and withered belly in a characteristic manner. Close observation will now usually detect, sometimes only at long intervals, peristaltic waves in the stomach, commencing in the left hypochondriac region, passing across the epigastrium, and culminating in the pyloric tumor which sometimes may be felt to become denser on contraction. At rare intervals a peristaltic wave may pass beyond the stenosed pylorus, and diffuse itself through the small intestines, but as a rule the visible contraction ceases at the pylorus. When this stage is well advanced, gastric dilatation commences, and may become excessive. It is recognized by the usual signs.

Tetany is a symptom noted in some cases.

Prognosis.—It is difficult to reach definite conclusions as to the prognosis of a disease about the pathology of which so little is known. If we follow some authors in placing every case of rebellious infantile vomiting in this category, the prognosis will be fairly good, since the largest proportion of such babies soon recover when their diet is regulated in quality and in quantity. If, on the other hand, we claim that all infants who recover without operation never had pyloric stenosis, or assert with Cautley and Dent that unless operated on all these patients die before they are four months of age—then, under these

circumstances, we repeat, the prognosis must be considered grave. And on our fundamental belief as to the gravity of the prognosis our ideas as to surgical treatment must be based. It is probably safe to assert with Meltzer that if these infants survive without operation more than four months their grade of stenosis must have been slight.[1]

Treatment.—It is needless to say that medical treatment always is tried first; but it is our belief that in the majority of cases medical treatment, no matter how promptly instituted and energetically applied will be unsuccessful in curing the patient. If the views as to the pathology of the affection set forth in these pages be correct, that the thickening is not neoplastic, but is developed as the result of irritation of some kind or another, then there is reason to think that medical treatment may in some cases be able to *prevent* the hypertrophic overgrowth of muscle tissue; but it is very doubtful if it is ever able to arrest it and to cause its disappearance after the hypertrophy has once developed. It is true that cures have resulted even in advanced cases, under judicious medical treatment; but they are extremely exceptional. Against the figures of Neurath (1899), who collected 41 cases of pyloric stenosis in infants less than twelve months old, all of whom died under medical treatment, may be opposed the best results of medical treatment as represented by the following reports: Walls reports nine cases treated without operation, with 3 deaths; Lowenburg informs us of a series of 14 cases treated without operation, with 5 deaths. Now in both of these recent series of cases it is to be presumed that operative treatment was available, even for the patients who died; and that among the patients who recovered without operation there were a number with such a slight degree of stenosis that operation need never have been seriously considered. Hence the inference is plain that taking any series of fully developed cases, the mortality following non-operative treatment is shockingly high. It remains for us to show what is the mortality following surgical treatment.

The most important fact to be learned from the surgical statistics published, is that *the earlier an operation is done, when once it has been determined upon, the greater is the chance of success.* Progressive loss of weight is the most imperative indication for operation. Unless weight is being lost it is almost certain that a sufficient amount of nourishment is being absorbed to sustain life, no matter how constant and copious the vomiting may seem to be. Fluoroscopy will show

[1] Four out of fifty patients under the care of H. C. Deaver (1920) recovered without operation; among the remainder, all of whom were operated on, there were four deaths.

how much of an ingested opaque meal is discharged through the stenosed pylorus; if at least two-thirds of the meal has passed through the pylorus within three hours, it is not likely that operation will be required.

When surgical intervention has been decided upon, it then becomes necessary to select some form of operation; and in doing this we should be guided not only by the change in the stomach, but also by the tender age of the patient. The operations employed oftenest have been *pyloroplasty* (and various modifications of this operation), and *gastro-jejunostomy*.

Bunts (1908) collected 69 cases of gastro-jejunostomy for infantile pyloric stenosis, with 37 deaths, a mortality of more than 53 per cent. Individual surgeons, however, especially Downes, Richter and Scudder have had much better results.

The operation of choice we believe is *Rammstedt's method of pyloroplasty*. No other methods of pyloroplasty, even Nicoll's, has given such uniformly satisfactory results. Rammstedt's first operation (1912) was modelled on two experiences of Weber's, which had been reported in 1910. Weber, in 1908 and 1909, operating on babies with pyloric stenosis, and attempting to do an ordinary Heineke-Mikulicz pyloroplasty but without opening the mucosa of the stomach, found that the sutures would not hold in the friable tissues when he came to close his longitudinal pyloric incision in a transverse direction. He was content to cover the pouting mucosa with an omental graft. Both his patients recovered. Rammstedt did the same operation without making any attempt to suture his longitudinal incision in the pylorus, and he left the unopened mucosa pouting into the gap. His patient likewise recovered. This, it will be seen is an even simpler form of pyloroplasty than that adopted by Nicoll, which, according to statistics published in the first edition of this work, was attended up to that time with the lowest mortality (15.38 per cent.) of any operation for infantile stenosis of the pylorus.

Nicoll's pyloroplasty is done as follows: An incision is made in A or V-shape (transverse to the long axis of the pylorus) down to the mucosa, which is not opened. The pylorus is then forcibly divulsed by forceps introduced through a separate incision in the anterior wall of the stomach. The incision in the pylorus is then closed so as to make a A or Y-shaped scar, thus increasing its breadth at the expense of its length. It is probable that the divulsion of the pylorus is unnecessary. Nicoll (1906) reported 13 operations with only 2 deaths.

Rammstedt's pyloroplasty is done as follows: A longitudinal incision is made in the anterior upper wall of the pylorus, in the least vascular

GASTROENTEROSTOMY FOR INFANTILE STENOSIS OF THE PYLORUS

Operator	Operations	Deaths	Mortality per cent.
Deaver (1920)[1]	12	2	16.6
Downes (1916)	31	11[2]	35.0
Richter (1914)	19	2	10.5
Scudder (1914)	17	3	17.6

RAMMSTEDT'S PYLOROPLASTY FOR INFANTILE STENOSIS OF THE PYLORUS

Operator	Operations	Deaths	Mortality per cent.
Deaver (1920)[1]	46	4	8.7
Downes (1920)	175	30	17.1
Gallie and Robertson (1917)	16	5	31.0
Gray and Pirrie (1919)	17	7	41.0
Mixter (1916, in Binnie's Operative Surgery)	8	1	12.5
Strauss (1918)	65[3]	3	4.6

[1] Eight of the gastroenterostomies and all of the Rammstedt operations were by Dr. H. C. Deaver. There was a series of 18 Rammstedt operations without a death.

[2] Three patients moribund before operation.

[3] Strauss says 24 of these patients were moribund before operation.

region, extending from healthy stomach wall past the pyloric tumor (Fig. 38). As the tumor stops abruptly at the duodenal end great care is necessary not to open the mucosa in the latter situation. The incision is carried down carefully through the muscular coats until the mucosa pouts into the wound. It is permissible to use the handle of the scalpel or the blunt ends of scissors (Fig. 39) to detach the muscular walls slightly from the underlying mucosa, but it is better not to attempt too much. Strauss (1915) uses only the handle of the scalpel after incising the serous coat of the pylorus. The incised muscular walls gape widely, and the mucous membrane pouts into the incision. Nothing more is done except to close the abdominal wall, the incision in which need not exceed 4 cm. in length. The operation takes from ten to fifteen minutes. The baby should lie on a hot water bottle, should be well covered, and the abdominal wall should be prepared before ether is given Haggard (1918) employs only local anesthesia. Strauss (1918) has modified this operation by shelling out two flaps of muscular tissue from the tumor and suturing these over the redundant and prolapsed mucous membrane (Fig. 40).

It is perhaps needless to add that most of the deaths following this

simple form of operation have been due not to the shock of operation, nor to its after effects, but to the fact that it was employed too late. In one of Downes's fatal cases the cause of the pyloric obstruction which had not been relieved by operation was found at autopsy to be a mucous polyp which blocked the canal like a ball-valve. This was not a true case of infantile stenosis of the pylorus, and there was no true muscular hypertrophy. Among 19 cases in which Downes added divulsion of the pylorus (as in Nicoll's operation) to the simple Ramm-

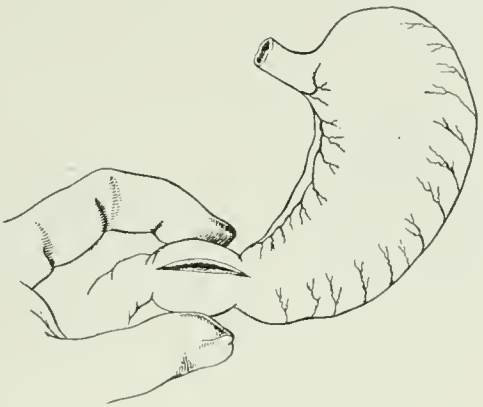

Fig. 38.

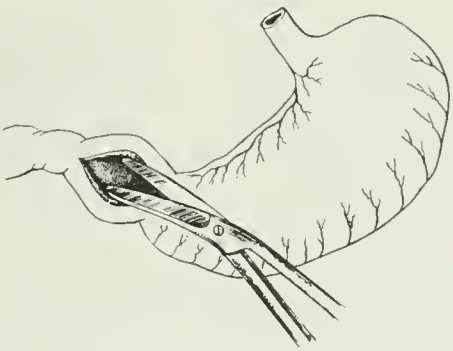

Fig. 39.

Figs. 38 and 39.—Rammstedt's Method of Pyloroplasty.

stedt procedure, there were two deaths from peritonitis. He has now abandoned divulsion and finds the results quite as good, without the risk of peritonitis.

In his latest statistics, which include a number of operations by Matthews, he has counted as operative deaths all patients dying in the hospital even though several died from gastroenteritis two to four weeks after the operation.

Several surgeons have had an opportunity to examine the pylorus some weeks or months after operation, the patient having died of

intercurrent disease. Thus Ransohoff and Wooley (1917) found six months after operation that the tumor mass had disappeared, and that the site of the operative incision in the pylorus presented a linear scar. In Gallie and Robertson's patient (1917), examination six weeks after operation showed no change in size of the tumor; while histological study of the specimen showed that the peritoneal covering of the pylorus had grown down into the operative incision, meeting on the exposed mucosa. Separating the lumen of the pylorus from the peritoneal cavity at this point was the mucosa, submucous areolar tissue, muscularis mucosæ, a small quantity of white fibrous tissue, and peri-

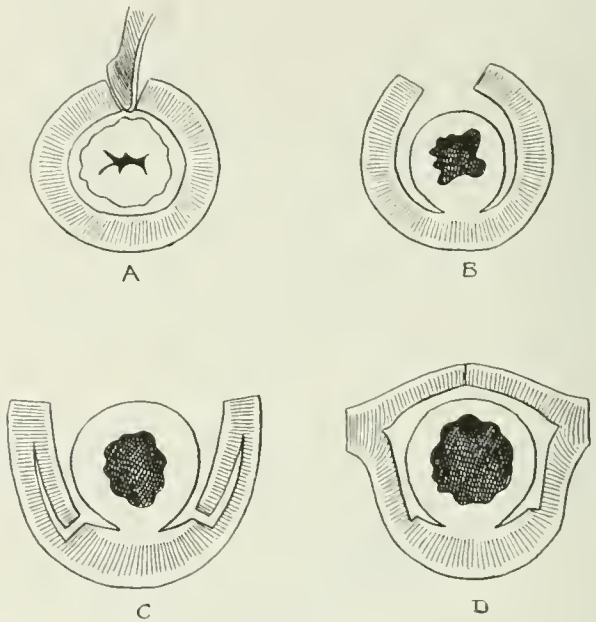

FIG. 40.—Strauss's Method of Pyloroplasty: A, after Incising the Serous Coat, the Muscle is Separated down to the Mucosa by the Handle of the Scalpel. B, The Muscle is Separated from the Mucosa around almost the entire Circumference. C, Flaps are Formed from the Hypertrophied Muscle Tissue, and D, are Sutured over the Mucous Channel which Expands with Obliteration of its Folds.

toneum. In another patient, also, who died of intercurrent disease about one year after the operation they found that the pyloric enlargement persisted, though its relative size compared to the normal intestines, was somewhat smaller than at the time of operation. Downes (1920) reports that autopsies on two patients who died of pulmonary conditions 18 months after leaving the hospital showed that the pyloric tumors had entirely disappeared.

Gastro-jejunostomy appears to be no more certain to bring relief, and is undoubtedly a more formidable undertaking in infants than is

Rammstedt's method of pyloroplasty; and we believe at the present day the surgeon must be bold indeed to employ the former method.

Eleven of the patients operated upon by the senior author, or his brother H. C. Deaver, have been traced a sufficient time after operation to be reckoned as end results: all were entirely relieved.

Pylorospasm

This is an intermittent or constant contraction of the pyloric sphincter, attended by more or less evident symptoms. It is really only a symptom of some other malady, or of one of a number of diseases met with in the abdomen. It will be noted that in the definition of pylorospasm it is stated to be an *intermittent* or *constant* contraction of the sphincter. It is very rarely a *remittent* contraction: that is to say, the spasm may occur only once or twice in a person's lifetime—it may be a spasm which "comes back at times;" or it may be, and more frequently is, an intermittent contraction—one which "goes away at times;" and in rare instances the spasm may be constant for a period of two or three days or longer, without any intermission. The first form is that which is a frequent accompaniment of gall-stone colic; and may occur in other acute abdominal affections, as appendicitis, being, here, as under other circumstances, merely a symptom of an organic lesion of the alimentary canal.

In many cases the pain of the cramp is not very great, amounting merely to a lively sense of discomfort in the epigastric region, and being overshadowed by symptoms of "peristaltic unrest of the stomach," so graphically described by Kussmaul (1880) ("embarras gastrique" of the French). When the pylorus contracts spasmodically, from whatever source of irritation there may be present, the stomach meets with an insuperable obstacle to its evacuation. Peristaltic unrest ensues, flatulence develops from fermentation and from swallowed air, and finally, when the limit of endurance is reached, the pylorus relaxes and gastric contents pass out into the duodenum, or the patient is relieved of his distress by vomiting, and comparative comfort is restored.

Such a crisis as this may occur, as already remarked, only once or twice in a patient's lifetime; or it may be the habitual sequel to every meal. The symptoms may vary from those of the mildest gastric indigestion, to the most awful and overwhelming pain in the region of the pylorus. The pain is a symptom of disease somewhere in the course of the mid-gut or its appendages.

It is not always possible to determine just what is the underlying disease in cases of pylorospasm. It is probably more often due to an erosion or ulcer of the stomach or pyloric antrum than to any other single affection; but as already remarked, it is a frequent accompaniment of affections of the gall-bladder and vermiform appendix; and may be the only distinctive symptom in patients with polypus of the stomach. Until further investigation teaches us more, we must be content in the majority of cases to treat the condition empirically.

If the pylorospasm persists, intermittently, over a long period, it is wont to be accompanied by symptoms of Reichmann's disease—excessive secretion of the stomach, gastro-succorrhea; but whether this disease is a sequel or a cause of pylorospasm physicians are not agreed; and a further discussion of the subject would be out of place in a work of this kind. Gastric dilatation may also follow; and it would not be improbable that hypertrophy of the pyloric sphincter might be a sequel of long standing pylorospasm in some patients, though we are not aware that such a change has ever been demonstrated.

Pylorospasm can be certainly detected by fluoroscopy after the ingestion of an opaque meal; but even when the diagnosis is thus decided, the cause may remain undetected.

Pylorospasm should first be treated energetically by medical means; and in cases where reasonable persistence along this line fails, surgical intervention must be considered. In all cases the condition of the appendix, as well as of the upper abdominal organs, should be investigated and appropriate operative treatment should be adopted.

Gastric Dilatation

Although the interest of the surgeon in gastric dilatation is usually confined to those forms which are strictly secondary in origin, and which are in the immense majority of cases, if not in all, produced by pyloric obstruction; and although for this reason it has seemed best to dicuss the subject under the general heading of obstruction of the pylorus, yet there are certain forms of dilatation of the stomach which have of late years come within the surgical horizon, and yet which are not technically due to obstruction of the pylorus. We refer to acute dilatation of the stomach, and to the form known to physicians as atonic dilatation or gastric myasthenia.

Acute Dilatation of the Stomach, first brought to the attention of the medical world in 1872 by Hilton Fagge, has been discussed in elabo-

rate articles by Neck (1905), by Conner (1907) and by Laffer (1908), the last named having collected 217 cases; Ruth has more recently (1913) collected 100 cases, which he has analyzed in a most instructive paper.

It is, as already remarked, primarily a medical disease. It is met with as a complication in various infectious diseases, such as pneumonia and typhoid fever, but occasionally seems to arise as a primary affection, the patient being suddenly seized with symptoms of obstruction, and there being no preceding disease of any kind. More often, however, and this is what has drawn surgical attention to it, the dilatation develops as a post-operative complication, and in many cases terminates fatally within twenty-four to forty-eight hours. The preceding operation is by no means always an abdominal one. Operations on the extremities, on the kidneys, and on other parts of the body have been followed by acute dilatation of the stomach; but a large number of post-operative cases have followed operations on the biliary tract.

Causes.—Various theories have been advanced to explain the condition, and their number shows that no one can be considered wholly sufficient to explain its development under the different circumstances in which it is encountered. As predisposing causes have been recognized: atonic dilatation of the stomach; pyloric obstruction whether associated or not with chronic gastrectasis; overfilling of the stomach with food or drink (lemonade in some and champagne in others have been accused as exciting causes); preexisting toxemias (typhoid fever, pneumonia, etc.); and lastly surgical operations. Routier (1905) apparently thought all post-operative cases could be attributed to septic intoxication; but while it must be acknowledged that in most instances when the condition is of long duration pronounced toxemia is present there have been very many patients with no evidence of sepsis. Ruth attributes the condition to *toxic paresis* of the stomach and upper bowels, often predisposed to by long anesthetization and operative trauma. Most patients are between twenty and thirty years of age. Zade adds to the predisposing causes already mentioned, that of abnormal length of the mesentery, or a position of the small bowels in the pelvis, thus pulling on the mesentery. Indeed the theory proposed by Hanau-Albrecht in 1899, that acute gastric dilatation is due to constriction of the duodenum by the superior mesenteric artery, through dragging on the root of the mesentery, still receives support. But most authors think that the dilatation is primary, and that it is merely increased by kinking of the pylorus or by the distended stomach itself pressing on and occluding the duodenum. The observations of Kelling

and others, referred to in Chapter II, as to the gastro-duodenal reflex, by which evacuation of the stomach is prevented by distention of the duodenum, have probably a close bearing on this subject; and since in many cases which have come to autopsy there has been found (Neck, 1905) some obstruction to the duodenum at its junction with the jejunum, and but rarely has there been found pyloric obstruction, it seems only fair to conclude that the mechanical obstruction thus produced is at least as sufficient an explanation as is the assertion that the dilatation is primary, or due to some lesion of the pneumogastric nerves, as suggested by Carrion and Hallon (1895). Laffer also supported this theory. It is not improbable, we admit, that in those cases of acute gastric dilatation developing after operations on the biliary tract, there may have been produced some reflex disturbances of gastric innervation by way of the splanchnics and the hepatic plexus; yet we are totally unable to see how a similar explanation could by any stretch of the imagination be considered applicable to the cases of those patients who had had operations performed on their lower extremitites. It appears to us that it is a much more likely thing that the anesthetization, and the lifting of the patient on and off the stretcher and the operating table, added to the frequently unusual and strained positions in which patients lie during and after operation, are all factors which would tend to produce an enteroptosis of the small intestines, or would in some way produce a kink at the duodeno-jejunal flexure, and so would be productive of the state of affairs usually found in connection with acute gastric dilatation Added to these causes, which might be present in every post-operative case, would be the direct interference with the viscera in abdominal operations of all kinds. Especially would this be the case in operations on the bile passages, where the duodenum and small intestines are constantly pressed by gauze pads out of their normal relations; and in operations for the removal of large ovarian cysts or myomatous uteres, where the small intestines would naturally fall into the emptied pelvis and occupy a position which in that individual patient would be strange and unusual. This theory was ably supported by P. Müller (1900). Seelig (1907) suggested that the application of a very tight abdominal binder might favor the occurrence of acute gastric dilatation, because although the small intestines may work their way by peristalsis down into the pelvis under such an obstruction, they will be unable to get back again, and as they accumulate in the lower abdomen will render the root of the mesentery taut.

Of the 102 cases analyzed by Conner, 42 (41 per cent.) followed

operations in which general anesthesia was employed (15 operations on gall-bladder, etc.; 17 after other abdominal operations; 10 after operations not involving the abdomen); other cases were observed during or after severe diseases (typhoid fever, pneumonia, etc.); others after injuries; others after indiscretions in diet; six were associated with disease or deformity of the spine, and four appeared to be idiopathic in origin. One patient with typhoid fever, who died from acute dilatation of the stomach, has come under the notice of one of us (Ashhurst) at the Episcopal Hospital, in the service of Dr. Charles H. Weber.

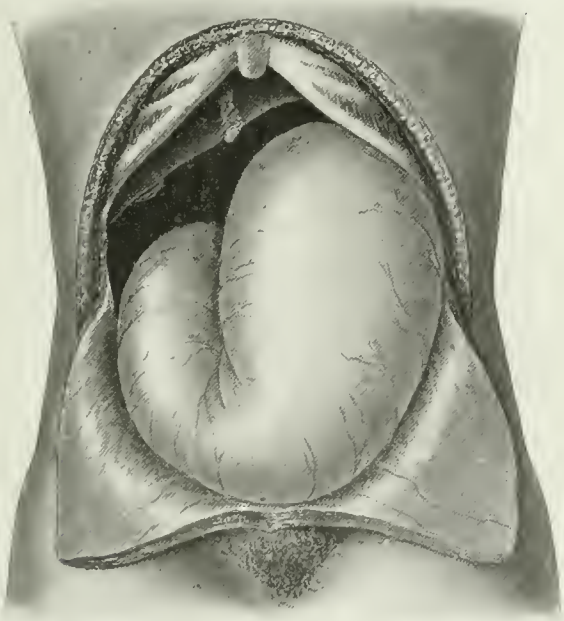

FIG. 41.—Campbell Thompson's Case of Acute Dilatation of the Stomach.

Pathology.—The stomach is found to fill practically the whole abdomen. Its shape is characteristic, presenting usually a marked V-shaped depression in the lesser curvature, and approximating the form found in gastroptosis, especially that due to deformity from tight lacing, though very much more pronounced. As pointed out in the previous paragraphs, a site of obstruction has most frequently been found in the neighborhood of the duodeno-jejunal flexure, or else where the superior mesenteric artery crosses the duodenum. Among 120 cases which came to autopsy, Laffer states that there was obstruction of the duodenum by the root of the mesentery in 27. The duodenum as far as the point of constriction is frequently much dilated.

There is rarely any obstruction at the pylorus, unless it is manifestly due to a kink produced by the descent of the stomach. The fluid contents of the stomach are due to hypersecretion, as well as to mere accumulation from obstruction of its outlets. But the presence of the immense quantities of gas, and its rapid re-accumulation after lavage are not so easily explained. The gas is no doubt in large part due to fermentation, and some of it is swallowed, as in the nervous affection known as aërophagia. But to account for the re-accumulation of gas in so short a space of time, some authors have held that gas was produced by transudation from the blood vessels in the stomach walls. Whether this is in accord with modern physiological views we do not know, but it appears to be a far-fetched explanation. Gas-producing bacteria have been found in the stomach contents, according to some reports.

There is as a rule no peritonitis; and the site of operation, if an abdominal operation has been performed, usually presents no deviations from the normal. Some writers have confused tympanitic distension of the stomach from peritonitis, with acute dilatation of the stomach. In the former condition, which is by no means so rare as acute dilatation, the whole intestinal tract may be much dilated, but the stomach, being the largest hollow organ, is apparently disproportionately dilated; hence the confusion.

Symptoms.—The symptoms of this malady cannot be said to be always very distinct or readily recognized. Usually the onset is sudden in character, and may begin from twelve to twenty-four hours after the operation, although in many instances no symptoms have developed until convalescence was thought to be assured. The patient's first complaint is generally *discomfort*, referred to the epigastrium, with a sense of distention. *Vomiting* occurs promptly, with comparatively little nausea; and large amounts of dark greenish fluid are gulped up without straining. The vomitus is almost never fecal, and is seldom very offensive. Bile and blood may be present, but usually the vomited matters are composed chiefly of gastric secretion and mucus, and present a characteristic smell. Vomiting generally persists to the end, whether this be the death or the recovery of the patient.

The *distention* of the abdomen may be readily recognized by the eye, being most prominent to the left of the median line. When the stomach tube is passed there is an abundant escape of odorless gas, with a gushing or a gurgling sound, at times almost an explosion; and a marked flattening of the abdomen usually follows evacuation of the

stomach by this means. But within a very short time after the stomach has been emptied it refills again, with secretion and air, and the patient's distress is as great as ever. It is extremely rare for any peristalsis to be observed. Palpation finds the abdominal walls not rigid, as in peritonitis, but merely tense from the tension within. A *splashing sound* is readily obtained from the stomach contents, and is usually too distinct for there to be any doubt that it comes from an air-containing cavity. The distention of the stomach is extreme, the greater curvature always reaching well below the umbilicus, and frequently extending to the pelvis. Percussion detects a *tympanitic note* over the most prominent portion of the distended stomach, and the usual change of level in the *dulness* produced by fluid can be obtained by turning the patient to one side.

Along with these local signs, the *general condition* of the patient is seen to have taken a sudden turn for the worse. There is usually little fever, but the pulse becomes thready and rapid, the eyes sunken and bright, the tongue heavily coated, the breath foul, the mind delirious or comatose, and dissolution appears imminent. In short the evidences of toxemia are pronounced.

In milder cases of the same nature, the stomach is relieved by lavage or by vomiting; and occasionally a profuse diarrhea is the first symptom that the obstruction has been overcome. In Roussel's patient (1908) there were from 25 to 35 extremely offensive movements daily. It is much better to remove the secretions by lavage, since sometimes fatal absorption from the small intestines will kill a patient in whom the subsidence of the dilatation of the stomach and the evident onward passage of its contents had given rise to hopes of recovery.

Differential Diagnosis.—It is important that the surgeon should not mistake the vomiting caused by acute dilatation of the stomach for that due to the anesthetic. In the former the symptoms usually do not arise until all nausea from the anesthetic has subsided; but occasionally when the post-operative nausea is severe and long continued the condition may pass into that of acute gastric dilatation without any pronounced change in symptoms. Peritonitis is frequently thought of when the symptoms of acute gastric dilatation commence. The period of onset of both frequently is similar, but the signs are not the same. Not only is the pain of a different character, being rather burning than sharp; but the physical evidences of a large amount of fluid, and above all the presence of the succussion splash, will at once show that peritonitis alone is not the condition present. Furthermore, the evacuation of the gas and other stomach contents with the sub-

sidence of the abdominal distention, which follow the passage of the stomach tube, confirm the diagnosis, and usually, for a time at least, produce a remission of symptoms. Neither peritonitis nor intestinal obstruction will be so affected. Finally, the nature of the preceding operation, or the previous course of the disease when no operation has been performed, may be sufficient to exclude both peritonitis and intestinal obstruction. Thus operations on the kidneys or the extremities, and even many abdominal operations, such as those for the radical cure of hernia, could under no normal circumstances be productive of peritonitis.

Prognosis.—Among the 217 cases of this affection collected by Laffer, 135 patients died, some within a few hours; but a few survived into the second week. One lived thirteen days. Seventy-seven patients are known to have recovered; and in 5 the result is not recorded. In Ruth's more recent series of 100 cases, there were 39 deaths. Any disease with such a death rate must be considered extremely grave.

Treatment.—As in other affections whose pathology is not well understood, so in acute dilatation of the stomach, treatment must be largely empirical. Lavage of the stomach is the first indication. As Terrier (1905) said, it is only a matter of common sense to empty an over-distended stomach.

In case acute dilatation of the stomach is not promptly relieved by the first lavage, not only should this treatment be repeated as often as indicated, but the patient should be made to lie on the left side, with the foot of the bed raised so as to bring the pelvis higher than the diaphragm. This failing to secure relief, the belly position may be tried, the patient lying prone in bed after the stomach has been emptied by the tube. In persistent cases, and where the nature of any preceding operation does not contraindicate it, the patient should be made to assume the knee-chest posture for fifteen minutes out of every two hours. When all these measures prove repeatedly futile, the abdomen should be opened, and the stomach, if still distended, should be emptied through an esophageal tube. If now a kink at the pylorus or at the duodeno-jejunal juncture be found and if it can be relieved without further interference, the surgeon should content himself with that; but in most of the reported cases it has been evident that no such simple procedure would have been productive of benefit. The surgeon should then adopt gastro-jejunostomy. The operation proposed by Robinson (1900)—section of the duodenum and its reunion in front of the superior mesenteric vessels,—is, as said by Finney, a thoroughly unpractical procedure.

Operative treatment of acute gastric dilatation appears to have been adopted in about 32 cases; among this number to which we have references, there were 17 deaths, a mortality of 53 per cent. Nearly all of these patients had been subjected to non-operative treatment without success, and this fact should be remembered in comparing the mortality of operative treatment with that of medical treatment. Moreover, it should be noted that whereas only 2 out of 14 operations (a mortality of 85.7 per cent.) recorded in the first edition of this work proved successful in saving the patients' lives, among 18 operations since recorded 13 proved successful (a mortality of 27.7 per cent.); showing that earlier recognition of the affection and more efficient surgical treatment are now able to save a considerable proportion of patients.

Among the cases analyzed by Ruth, the gastric tube, with or without lavage proved successful in 50 out of 71 cases in which this method is known to have been employed; of 11 cases in which the stomach tube is known not to have been used, 7 proved fatal.

Operations for Acute Dilatation of the Stomach

I. Exploratory Laparotomy. 13 cases with 7 deaths; mortality 53.8 per cent.
- Abdomen closed without emptying stomach:
  - Jessop (Lancet, 1888, i, 726). Death.
  - Robinson (Cincinnati Lancet Clinic, 1900, xlv, 577). Death.
- Stomach emptied itself:
  - Lanphear (cited by Ruth: Am. J. Obst., 1913, lxviii, 525). Rec.
  - Templeton (Clin. Jour., 1909, xxiv, 302). Rec.
- Stomach emptied by tube passed through esophagus:
  - Littig (cited by Ruth: Am. J. Obst., 1913, lxviii, 525) Death.
  - Macevitt (N. Y. State J. of Med., 1906, vi, 284). Rec.
  - Moorhead (J. A. M. A., 1909, lii, 1909) Rec.
  - Turner (Appendicitis, Hernia, and Gastric Ulcer. London, 1905, p. 113). Death.
- Adhesions or kinks released:
  - Axhausen (cited by Ruth: Am. J. Obst., 1913, lxviii, 525). Rec.
  - Borchgrevink (Surg., Gyn. and Obst., 1913, xvi, 662). Death.
  - Linke (Beitr. z. klin. Chir., 1914, xciii, 360). Death.
  - Petit (Thèse de Paris, 1900; cited by Conner: Am. J. Med. Sc., 1907, i, 345). Recovery (after jejunopexy).
- Volvulus of intestine reduced, dilatation of stomach not found at operation:
  - Lichtenstein (Zentr. f. Gyn., 1906, No. 44). Death.

II. Gastrotomy. 8 cases with 5 deaths.
- Appel (Phila. Med. J., 1899, iv, 314). Death.
- Box and Wallace (Lancet, 1898, i, 1538). Death.
- Farquhar (Brit. Med. J., 1911, i, 675). Rec.
- Finney (Bost. Med. and Surg. J., 1907, clv, 107). Death.
- Hansen (cited by Borchgrevnik: Surg., Gyn. and Obst., 1913, xvi, 662). Rec.
- Hoffman (Münch. med. Woch., 1904, li, 2003). Death.

MacMonagle (cited by Ruth: Am. J. Obst., 1913, lxviii, 525). Recovery after emptying stomach through trocar.
Wright (Practitioner, 1897, vi, 598. Death.

III. Gastrostomy. 2 cases, with 1 death.
Brown (Lancet, 1899, ii, 1017). Death. (Stomach mistaken for pancreatic cyst.)
Sommarin (cited by Ruth: Am. J. Obst., 1913, lxviii, 525). Rec.

IV. Gastrojejunostomy. 9 cases with 4 deaths.
Baillet (Bull. Soc. Chir., Paris, 1909, xxv, 326). Death.
Lanz (Nederl. Tijdschr. v. Geneesk., 1913, lvii, 279). Rec.
Lanz (Ibid.). Recov.
Linke (Beitr. z. klin. Chir., 1914, xciii, 360). Death.
Linke (Ibid.). Recov.
Kehr (Arch. f. klin. Chir., 1897, lviii, 632). Death.
Körte (Deutsch. med. Woch, 1904, xxx, 1554). Death.
Stierlin (Corresp.-Bl. f. Schweiz. Aerzte, 1913, xliii, 1089). Rec.
Torrance (N. Y. Med. J., 1909, lxxxix, 70). Rec.

Atonic Dilatation of the stomach, or **Gastric Myasthenia,** though formerly a well recognized affection of the stomach, has of late years become a disease of the utmost rarity. This is largely due to the increased accuracy in diagnosis of gastric affections to which physicians have attained, but in no small measure is it due to modern surgery which has proved by the living pathology of the operating table that most of the cases formerly classed as atonic dilatation are really examples of gastric dilatation due to well defined lesions, chiefly to pyloric obstruction from carcinoma, ulceration or perigastric adhesions. There is no doubt that after such debilitating diseases as typhoid fever the gastric walls may become weakened, and become readily subject to distention and dilatation when overloaded; but even in cases such as these, there is no good reason to suppose that recovery, if not attained by medical measures, may not be aided by operative means. Long-standing gastritis, originally catarrhal in form, may eventually invade the submucosa, thickening and hardening the gastric walls, and thus materially interfering with peristalsis. In the same manner, but even more noticeably, ulcerations, cancerous growths, and even the ingestion of poisons, may greatly impair gastric motility without in any way producing stenosis of the pylorus. It is not impossible that the gastric nerves may be the seat of disease, without there being any change in the gastric wall itself, and that by this means dilatation may ensue from loss of motility. But such a change is probably much rarer than it has heretofore been considered. Actual degenerative changes (colloid, fatty, etc.) in the muscle fibres of the gastric walls are probably of less infrequent occurrence.

In patients with this form of gastric dilatation, the absence of distinct history is the chief means of differentiating the disease from that form due to pyloric stenosis. The symptoms from which the patient seeks relief are the same in kind, though probably less in degree, than in pyloric stenosis. The sense of fullness persisting from one meal to the next, the anorexia, the thirst, the gaseous distention, and the eructation—all are the same in both affections.

Three stages of gastric myasthenia may be recognized. The first is **the stage of compensation,** the second that of **stagnation,** and the third that of **retention.** Early in the disease the symptoms are not of such prominence as to fix themselves in the patient's mind; it is only after an unusually heavy meal, or after a particularly indigestible one, that he is made aware of his dyspepsia. The gaseous distention then becomes oppressive, the clothing is perhaps unconsciously loosened, and relief is eventually obtained by the belching of gas, or by the lazy emptying of the wearied stomach into the duodenum. Not unfrequently a little sour fluid rises into the mouth along with the gas. But in this stage compensation is generally sufficient, and these periods of broken compensation arise only when some unusual strain is thrown upon the stomach. This stage may last for months or years; but it is exceedingly prone to pass into the second stage—that of gastric stagnation, a condition in which the stomach is unable completely to evacuate its contents between meals, except between the evening meal and breakfast, an interval sufficiently great for evacuation to be accomplished. The patient finds it impossible to gain in weight, though it is not usual for weight to be lost. Digestion, though delayed, is eventually completed. When, however, the third stage, that of retention, is reached, emaciation commences and may become extreme. The stomach is not emptied even during the night, and lavage before breakfast will detect particles of food still in the stomach; and the gastric contents will possess the usual characteristics of retention—they will be sour, rancid, and usually very acid. Occasionally, when atrophy of the mucous membrane is present, the contents are neutral or alkaline in reaction. The evidences of fermentation are pronounced, and the production of gas will continue oftentimes after the stomach contents have been removed by lavage, as is evidenced by the separation of these contents into the usual three layers. The dilated stomach, by dragging on the pylorus, causes a kinking near the latter, thus adding the mechanical factor of actual obstruction to the myasthenia which was the primary cause of the dilatation.

Secondary nervous symptoms are of common occurrence in patients suffering from gastric retention due to atonic dilatation. The source of these symptoms is to be found in intestinal toxemia. The intestines partake of the atony which affects the stomach, and it is precisely because there is no pyloric obstruction that the fermenting gastric contents in part reach the small bowels, and are thence absorbed. Where the gastric dilatation is due merely to mechanical obstruction at the pylorus, this obstruction itself protects the small bowels from the decaying food, and the various symptoms of hypochondriasis, hallucinations, dyspnea, tachycardia, urticaria, erythema fugax, and other toxemic affections are the exception rather than the rule.

Treatment.—The treatment of gastric myasthenia in its earlier stages should be medical. If the first stage be recognized it often times may be cured, or the development of the second may at least be indefinitely postponed, by regulation of the diet and tonic treatment. During the second stage, in addition to the above, lavage is indicated; and electricity may be employed with some hope of benefit. But when once the stage is reached where weight is progressively lost and where relative pyloric obstruction is present (either from kinking or from relative stenosis of this orifice of the stomach), then purely medical measures no longer will be found efficient. It is rarely possible by medical means even to keep the patient from losing more ground, let alone improving him. In such cases as these we think operation should be undertaken, provided no contraindication to any operation exists; but the patient must not be led to expect an immediate cure. Probably the most that surgery can do is to so alter the mechanics of the stomach and intestines that medical measures will become effective. Hence it is to be anticipated that a prolonged course of medical treatment will have to be carried out after the operation has been performed.

As to the special form of operation to be employed, it has long been the consensus of opinion that gastro-jejunostomy is the best. We feel however, that surgeons now more generally adopt Finney's pyloroplasty either alone, or combined with some form of gastroplication. It is difficult to decide upon the respective merits of these two methods, because, as already remarked, we do not anticipate the phenomenally rapid amelioration of symptoms after operation for this condition that we do in cases of pyloric obstruction without marked atony; and too many medical men are inclined to give credit for the slowly acquired improvement solely to the medical treatment employed,

when there can be no doubt, at least so it seems to us, that without the operation which improved the receptivity of the gastro-intestinal tract, food and drugs would have been of as little avail as they were before the patient was brought to the surgeon. While our own preference in the past has been for gastro-jejunostomy in such cases, as far as we can judge surgical opinion at present, it is in favor of plastic operations on the pylorus for these patients, and is opposed to gastro-jejunostomy for any but obstructive cases.

Secondary Gastric Dilatation.—In dealing with this condition the surgeon must never lose sight of the fact that it is not a distinct disease. Ever before his eyes must be the picture of a stomach that has become dilated after ineffectual efforts to overcome an obstruction to its evacuation. Only in this way will he be able to appreciate the seriousness of that stage of the disease at which his unfortunate patient has arrived. Were medical treatment always effectual in treating the disease, even when instituted at the commencement of the malady, the surgeon would never see any patients with gastric dilatation. It is the terminal stage of a serious disease, and as such is the gravest stage. The patient may have been in danger from hemorrhage or from threatened perforation at earlier periods of his malady; but in addition to these dangers, which, though perhaps less imminent, still persist, he is now afflicted with the most serious complication of all, save that of carcinomatous degeneration. And gastric dilatation due to benign obstruction is less serious than gastric carcinoma only because patients with the forner disease die more slowly than do those with cancer. Without surgical relief, both diseases are equally fatal: cancer usually kills in a shorter time, but death in benign gastric dilatation is quite as sure even if longer delayed.

Causes.—Although gastric dilatation in the immense majority of cases is caused either by *carcinoma* or by *ulceration* about the pylorus, yet in exceptional instances other factors are operative, and should therefore be borne in mind. It is well to remember, also, that *changes in the duodenum*, similar to those occurring at the pylorus itself, are not infrequently productive of gastric dilatation. Especially is this true of ulceration and cicatrization above the ampulla of Vater; but ulcers even below this site, as well as other affections of the duodenum similar to those implicating the pylorus, may also cause dilatation of the stomach.

Systematic writers are in the habit of classifying the causes of obstruction here, as elsewhere in the alimentary tract, as those from changes in the wall of the pylorus, those within the lumen of the canal,

and those which cause obstruction by distortion or pressure from without. Among the changes in the pyloric wall itself, the development of carcinoma probably holds first place in the production of gastric dilatation. It will be more fully considered in a subsequent chapter. Next to carcinoma, pyloric obstruction, and consequently gastric dilatation, is most frequently due to hyperplastic or cicatricial changes produced by benign ulceration. As will be presently pointed out, a temporary pyloric obstruction may be caused by hyperplastic ulceration at the pylorus, and later in the course of the ulcer's evolution the hyperplasia may subside, and the pylorus again become patent for a short time. At this stage the symptoms of gastric ulcer may temporarily disappear, and the patient may consider himself cured. At a later date, however, the latent ulcer will again give evidence of its existence, when by its cicatricial contraction the pylorus again becomes obstructed, this time permanently. The earlier and temporary obstruction usually does not cause gastric dilatation; it is rather productive of increased peristalsis, with hypertrophy of the muscular walls, and is frequently accompanied by pylorospasm, gastrospasm, and peristaltic unrest of the stomach (see p. 141).

Apart from these two changes—carcinomatous and ulcerative—there are few others taking place within the walls of the gastro-duodenal canal which are ever productive of secondary dilatation of the stomach. Yet Moullin (1907) called particular attention to fibrosis of the pylorus without evidence of past or present ulceration, as a cause of stenosis; and similar cases still occasionally come under the surgeon's care. Causes of obstruction acting from within the gastro-duodenal canal are very rarely causes of gastric dilatation. The pylorus may be obstructed by foreign bodies, or by hair balls, or by concretions due to medicines such as bismuth administered in large quantities or over long periods of time; but it is extremely uuusual for such agents to cause any but intermittent obstruction of the pylorus. The same is true of such pathological changes as gastric polypus (see p. 210). Pylorospasm may be the only evidences of such changes.

But the agents are many which from without the alimentary canal may cause gastric dilatation by means of pyloric or duodenal obstruction. Not only may *adhesions* act in this manner, but a *distended gall bladder*, or a large *biliary* or *pancreatic calculus* may similarly be productive of dilatation of the stomach. The senior author has had a number of cases of pyloric and duodenal obstruction with secondary gastric dilatation following the removal of the gall bladder due to adhesions either of the pylorus or the duodenum to the site of removal of

the gall bladder, all of which were permanently relieved by posterior gastroenterostomy. To prevent such adhesions from forming after cholecystectomy, not only should the gall-bladder bed be most carefully repaired, but the great omentum should be drawn up and interposed between the liver and duodenum. *Chronic pancreatitis* and *cancer of the head of the pancreas* may so obstruct the duodenum as to cause secondary gastric dilatation. *Enlarged glands* in the portal fissure of the liver, or along the common bile-duct as well as retroperitoneal tumors, aneurisms, etc., may all in exceptional cases be productive of secondary gastric dilatation. Moreover, in addition to such causes, the *displacements* of the various abdominal organs may eventually lead to the same result. The influence exerted by a *floating kidney* is somewhat hypothetical in this respect; but there is no doubt that enlargement of a fixed right kidney may cause pyloric obstruction[1] and there is good evidence for believing that displacements of the *liver* due to tight lacing or other causes may be productive of dilatation of the stomach. The *modus operandi* of the change in the latter case is not always easy to detect; but it probably is either by directly obstructing the pylorus, or by first producing gastroptosis, which in turn brings about a kinking of the pylorus.

Perigastric adhesions—the result of perigastritis, as the condition was called when it was considered a distinct disease, analogous to perityphlitis—are due to a variety of causes. The adhesions, however, which are productive of gastric dilatation are usually to be traced to affections of the biliary tract. Indeed, disease of the biliary tract is in many instances the origin of the whole chain of gastric disorders. This connection has frequently been noted (page 452). In a smaller number of instances, perigastric adhesions causing pyloric obstruction have arisen in attacks of plastic peritonitis due to gastric ulcers themselves; and in exceptional cases are due to previous attacks of peritonitis from other causes. A glance at Figure 42 (Andrews) will show how disabling these adhesions may become.

Clinical Pathology.—The changes occurring in the pylorus and the stomach in secondary gastric dilatation are reflected with fair accuracy in the symptoms which are observed. In the early stages of gastric ulceration there frequently occurs such hyperplastic reaction as to cause obstruction of the pylorus, if not to produce a palpable tumor. Such inflammatory masses as these, producing pyloric obstruction, and simulating a malignant tumor, have on several occasions (see p. 263)

[1]The junior author had such a patient, with pyonephrosis, under his care in 1914, in the Episcopal Hospital.

led surgeons to the performance of gastro-jejunostomy, in the expectation that the patient would thus obtain some little respite from imminent death. In such cases great have been the surprise and the joy of the surgeon and the patient to observe after a few months that the suspected tumor has melted away, as it were, the relief obtained by means of the operation enabling nature to produce a cure of the disease. If no operation had been performed at that time, one of

FIG. 42.—Perigastric Adhesions, Involving Gall-bladder, Colon, and Sigmoid Flexure. The Small Intestine has been Cut Away. (*Andrews.*)

three courses might have been pursued by the disease. The first and most usual course, we think, would be that under appropriate medical treatment sufficient rest would have been obtained by the inflamed area for a decrease in the obstruction to have occurred, with a subsidence of the symptoms of peristaltic unrest of the stomach. This would be only a temporary lull, however, whereas after a gastro-jejunostomy we should have reason to expect a cure. The second course might have been for the obstruction to persist and to lead

without delay to gastric dilatation; while the third course, and one which we are satisfied is less rare than that just mentioned, would be for the hyperplastic area to undergo malignant change, whether or not a temporary lull occurred in the evolution of the disease under medical treatment.

If the obstruction of the pylorus, due to the hyperplastic process, decreased under medical treatment, the symptoms would naturally abate, and the patient, and the physician as well would naturally regard the disease as cured. But in the vast majority of such cases there comes a time, perhaps months or even years later, when the gastric ulcer in its course of cicatrization and contraction again narrows the pyloric orifice, and when the old stomach symptoms begin afresh. It is at this stage of the disease that secondary dilatations are most frequently seen.

When due attention is paid to the various causes of pyloric obstruction, it is not difficult to picture the course which the disease will pursue in each individual case. As a rule, a temporary gastric hypertrophy occurs, with increased peristalsis, and for a time the obstacle may be overcome. Sooner or later, however, the gastric walls yield, and from the stage of compensation that of stagnation is reached, and this finally drifts into absolute retention. The downhill course is most rapid in malignant pyloric obstruction; but with judicious medical treatment it may, in patients with benign obstruction, extend over a period of years.

Symptoms.—The symptoms of secondary dilatation of the stomach are usually sufficiently pronounced. In the earlier stages of the disease, when compensation is present, or even later, when stagnation has commenced, the symptoms are less distinct; but when once retention has developed, there is slight probability of making a wrong diagnosis.

The **subjective symptoms** are much the same as those which have already been described under atonic dilatation of the stomach. But the *previous clinical history* of the patient will usually throw much light upon the diagnosis. Usually the patient at an earlier date has presented symptoms of gastric or duodenal ulcer; and possibly there has occurred a lull in the evolution of the disease, when the ulcer was healing or had actually healed, and before it had contracted, or while the tone of the gastric walls was still sufficient to compensate for the slight obstruction present. After this temporary abeyance of symptoms there will gradually be developed the *sense of fullness* presisting after meals, perhaps even to the time of the next meal, and thus leading to *anorexia*.

Because fluids are not absorbed from the stomach, and because in the stages of stagnation and retention they are late in reaching the small intestine, if they reach it at all, there is more or less *constant thirst*. As the dilatation progresses, and as stagnation becomes extreme, the dilating stomach occasionally makes the attempt to empty itself by the act of *vomiting*. Generally it is an ineffectual attempt, some of the stomach contents not being expelled; but this partial evacuation procures an intermission in the nausea for a couple of days. The very fact that ingestion of food does not always provoke emesis shows that the ulceration has passed the irritable stage; and the copious and cumulative vomiting which recurs every second or third or fourth day is, in itself, very good evidence that the stomach is dilated.

To these usual symptoms should be added one less usual, but of increasingly frequent occurrence in these last years, when the pathology of gastric disorders has become better understood. We refer to *gastric tetany*, which may, we think, be most appropriately considered as a symptom of dilatation of the stomach. This was first described by Kussmaul in 1869. He thought it was caused by dehydration of the tissues, due to the pyloric obstruction. The theory of autointoxication, according to Fleig (1908), was first systematically put forward in 1881 by Reuss. But to this theory it may be objected that autointoxication is rare without intestinal putrefaction, and that in most cases of gastric tetany there is no evidence of intestinal putrefaction, the pylorus being obstructed and preventing the discharge of gastric contents into the intestinal canal whence alone they may be absorbed. We are rather inclined to assert with Fleig that autointoxication can at most be an accessory, but not the main, cause, which we believe is dehydration of the tissues. Possibly as Fleig suggests autointoxication and dehydration may alter the chemistry of the thyroids or parathyroids. In support of dehydration as a cause, Fleig calls attention (1) to the numerous case reports of gastric tetany where the existence of dehydration was undeniable, and where symptoms were relieved by the introduction of fluids; (2) to the occurrence of cramps more or less like those of tetany in many diseases where dehydration is a marked feature, such as cholera, typhoid fever, infantile diarrhea, etc.; and (3) to the production of cramps in the lower animals by artificial concentration of the blood by means of intravaneous injections of almost any hypertonic solution (sodium chloride, glucose). Cramps may even follow the polyuria or diarrhea produced by diuretics or purges. MacCallum and his associates have suggested (1920) that deprivation of the tissues of chlorin, owing to exclusion from the intestinal tract of the hydro-

chloric acid of the gastric juice, may be the cause of the twitching and convulsions. They point out that there develops an extreme increase in the alkali reserve of the blood, and that experimentally such symptoms may be *prevented* by constantly furnishing a large supply of chlorids. The *cure* of the condition they found by no means so easy.

It is needless to dwell here upon the symptoms of tetany, as they are detailed in every text-book of medicine; but it is worth while noting that certain of the so-called diagnostic signs may be absent. Trousseau's phenomenon (production of typical cramps by obstructing the venous circulation of the arm) was absent in cases reported by Albu, Ast, and Schlesinger. Chvostek's sign (production of cramps by the lightest tapping of the motor nerve trunk, especially the facial) probably is more constant. Hoffman's sign (slight pressure over sensory nerves causing paresthesia) is not very reliable. Erb's phenomenon (increase of galvanic and sometimes of faradic excitability of all nerves except the facial) is not often tested, nor is Schlesinger's phenomenon (painful supination of feet when the hip is flexed while the knee is kept extended). We do not believe that it is necessary for the surgeon to delay treatment until all these classical signs develop in full intensity. In mild and chronic cases this may be possible, but as ordinarily seen the condition is at the least subacute if not actually acute and the patient very ill. Under such circumstances it is sufficient for the surgeon to find a patient suffering with cramps or spasmodic twitchings in association with copious vomiting, suggestive of a dilated stomach, for him to institute active treatment. McKendrick (1907) referred to 63 cases of tetany which were clearly due to gastric dilatation. One fatal case of gastric tetany following gastro-jejunostomy with occlusion of the pylorus has come under the notice of one of us (Ashhurst)

William M., 44 years of age, was admitted to the Episcopal Hospital in November, 1915, complaining of vomiting of blood and pain in the epigastrium. Two years previously he had been operated on by another surgeon, gastro-jejunostomy being done for an ulcer on the lesser curvature near the cardia; there had been no pyloric obstruction, and the pylorus had not been occluded at the time of operation.

Operation by Dr. Ashhurst (Nov. 9, 1915). The omentum was adherent to the former incision. The pylorus was normal. There was a mass on the lesser curvature of the stomach extending about 8 cm. down from the cardia. There were enlarged, hard, discrete, lymph nodes in the gastro-hepatic omentum. The anterior wall of the stomach was opened parallel to its blood vessels, and the crater of a callous ulcer, just admitting the finger tip, was palpated in the centre of the indurated area. The tumor (presumably benign) extended so far toward the cardia that it was not considered safe to attempt its excision. The old gastro-jejunostomy opening would not admit the tip of the finger within the stomach. The incision in the stomach was then closed, and the great omentum and transverse colon delivered. By detaching for 5 cm. some avascular adhesions between the under layer of the mesocolon and the jejunum the site of the old anastomosis was exposed,

and it was now found permeable to the finger. To enlarge the anastomosis which was about 3 cm. in diameter the posterior gastric wall and the jejunum were incised in the long axis of the anastomosis for a distance of 5 cm. each, and the incision was reunited transversely. Finally the pylorus was obstructed by a linen purse-string suture supplemented by longitudinal infolding of the pyloric canal. It was hoped these measures might permit healing of the ulcer.

Nov. 15. The patient has done well and today was given soft diet. Later he vomited a litre or more of fluid all at one time. Liquid diet was resumed and enteroclysis of glucose and sodium bicarbonate solution was given.

Nov. 17. Slight rise of temperature (100.6° F.). Enteroclysis retained. Mind not clear: he is rather noisy and cries out as with pain. He makes peculiar motions with his arms, and his fingers seem rigid and partially contracted. The condition resembles the beginning of a spasm. Only cracked ice by mouth.

Nov. 20. Better. Liquid diet since yesterday without vomiting. Mind is not quite clear yet and tetany-like movements persist.

Nov. 21. Abdominal incision healed. General condition better. Pulse averages 100, respirations 24, and temperature remains normal.

Nov. 23. Slightly worse. Mouth dry. Still twitching of muscles and irregular movements of extremities.

Nov. 25. Slowly failing.

Nov. 26. Twitchings still present. No dilatation of stomach can be detected.

Nov. 27. Noisy and restless. Died at 11 A. M., eighteen days after operation. No autopsy was permitted.

Bircher (1911) reported one case of gastric tetany which followed 9 or 10 days after gastro-jejunostomy for duodenal ulcer. In neither of these cases did there seem much doubt that the condition was due to dehydration, from copious vomiting. Most cases on record have not followed any operation, but have arisen in patients with chronic gastric dilatation with retention. According to Bircher the mortality of the condition under medical treatment is 75 per cent. or higher. Kinnicutt (1909) succeeded in controlling the spasms by intravenous injections of soluble calcium salts (usually 4 to 1000) in one patient, but death eventually occurred; autopsy by Opie showed no lesions in the parathyroids. However, in three of Wirth's collected cases the parathyroids were found at autopsy to be diseased. According to Wirth's statistics (1910) operative treatment offers much better chance for recovery than does non-operative. He collected 21 cases of operative treatment, with only 3 deaths (15 per cent. mortality), 17 cures, and one patient improved. Bircher's second patient, who recovered after gastro-jejunostomy, is not included in Wirth's figures. Bircher advises for the worst cases that only jejunostomy be done, as the simplest and quickest way of relieving the pyloric obstruction; in others a pyloroplasty or gastro-jejunostomy is indicated.

According to some authorities, *Globus Hystericus* is frequently due to the drag on the esophagus exerted by a dilated or proptosed stomach.

The **objective symptoms** of secondary gastric dilatation are even more characteristic than are the subjective. The *capacity of the stomach* is seen to be increased, not only from the excessive amount of matter vomited, but from the amount of fluid that may be introduced through the stomach tube. Dilatation of the stomach with air will also make its great size apparent. In men the dilatation is more horizontal, while in women the increase in size is chiefly toward the pelvis. The level of the greater curvature is nearly always found below the umbilicus, and in women it not infrequently reaches to the symphysis pubis. The dilatation with air should be very gradually done by means of a hand bulb attached to the stomach tube. The stomach should meanwhile be lightly percussed and the sensations of the patient should be the infallible guide as to the limit of distention to be produced. While in most cases of open ulcer we think even the passage of a stomach tube should be avoided on account of the possibility of exciting hemorrhage or producing a perforation, in secondary dilatation of the stomach we think no damage can be done, provided common sense is exercised and the manipulations are carried out with gentleness and patience. Hurry should be avoided above all things; it is under such circumstances the equivalent of violence. The use of a Seidlitz powder, its separate parts administered at short intervals one after the other, may be more agreeable in anticipation to the patient, but it is a dangerous and uncontrollable remedy, and as such should be avoided. It is impossible to determine beforehand either the force of the effervescence or the capacity of the stomach; and while we are well aware that this means of distention has been employed many more times safely than with disaster (see p. 297) yet it is a method which in our opinion is barbaric in its simplicity. The outlines of the stomach may also be determined by means of *skiagraphy*, after the administration of an opaque meal.

The examination of the *contents of the stomach* reveals the usual fermentative and putrefactive changes of gastric retention. The fluid withdrawn settles into three layers—the lowest of semi-solid matter, the middle of clear or slightly cloudy yellow fluid, while the topmost layer is extremely frothy, due to the gas-producing ferments and micro-organisms.

The *feces* of the normal individual contain from 4 to 6 ounces of solid matter in twenty-four hours, and about 75 per cent. of water. As a result of the lessened absorption which occurs in gastric dilatation, the amount of solids decreases to one and a half or two and a half ounces, and the proportion of water falls as low as 40 or even 30 per cent.

The *urine* is also much diminished in quantity, and the amount of urea and chlorides is decreased.

Diagnosis and Differential Diagnosis.—As has already been mentioned, extreme degrees of gastric dilatation are seldom mistaken for other affections. It is in the early stages—those of compensation and mild stagnation—that the disease is most frequently overlooked. Such patients are classed as dyspeptics, and are treated in many medical dispensaries for chronic gastritis; the diagnosis is based on the symptoms alone, without any attempt being made to trace the evolution of the disease or to apply to it the principles of physical examination of the secretions, and their digestive power—methods of study which are nevertheless constantly employed in studying the kidneys, the cardiovascular system, and the lungs. It is in these early stages that the pathologist's findings from examination of the gastric contents may give the first clue as to the nature of the disease. But it is only a clue, and should be so regarded. Were these chronic dyspeptics studied with the care their sufferings merit, the dispensaries of some hospitals would have fewer return visits, but more patients would be permanently cured of their maladies by surgical means, before their strength and vitality had ebbed so low that scarcely with forced feeding and stimulation will many of them be brought to the condition where they may be considered good operative risks.

When the early stages of gastric dilatation are once recognized, it next becomes important to determine the cause of the dilatation; for as we have already seen, there exist two distinct groups of gastric dilatation, the atonic and the obstructive. The former is rare, and it is our belief that it grows rarer every year, as more patients are subjected to operation, and as the surgeon is given more opportunities to show that the disease is really of an obstructive nature.

With due attention to the previous history of the patient and strict inquiry into the clinical course of the present illness, of which illness gastric dilatation is a stage, it will in most instances be quite possible to draw a distinction between myasthenic and obstructive dilatation of the stomach. When the symptoms of gastric dilatation appear after a distinct period of gastric trouble, whether immediately or remotely preceding the present symptoms, obstruction is almost certainly the cause. If the dilatation has developed rapidly, in the course of a few weeks or months, without a long history of preceding gastric indigestion, especially if the patient be past early adult life, malignant disease is probable. When no preceding indigestion, typical of gastric or duodenal ulcer, or of biliary infection, has annoyed the

patient over a long period of time—when, in short, the clinical history is negative—then it is possible that we have to do with a case of myasthenic dilatation. A point in the differentiation of obstructive from atonic dilatation of the stomach, on which much stress has been laid by Van Valzah and Nisbet, is that in the former variety of dilatation solids are much more obstructed than are liquids; and on this account toxemic symptoms are less usual than in atonic dilatation, in which latter affection the fermenting stomach contents every now and again are discharged into the intestinal canal, whence they may be absorbed. In obstructive dilatation fluids are evacuated rapidly compared to the rate of evacuation of solids, so long as the stages of compensation and stagnation persist; and Van Valzah and Nisbet claim that in this disease (obstructive dilatation) if 500 cc. of water be given when the stomach is empty, it will be evacuated within one hour and a half, or long before the atonic (myasthenic) stomach "ceases to splash or to yield water upon the introduction of the tube." Atonic dilatation, they remark, was once called the "dyspepsia of liquids."

In myasthenia pain and vomiting are exceptional; in obstruction pain is a prominent feature, especially when perigastric adhesions exist, and copious vomiting every few days is the rule. Finally, myasthenic dilatation is usually considerably relieved within a reasonable time by medical treatment, while the obstructive form grows progressively worse, even when such treatment is instituted in the early stages.

Dilatation of the stomach must not be confounded with a *simple large stomach*, whether it be congenitally of an abnormally large size, or due to long continued overfilling. In such a stomach an attack of gastritis, due to some unusual indiscretion in eating or drinking, may simulate for a time gastric dilatation. But the inflammation in such cases is quickly relieved by functional rest and medical treatment, which is not the case where the stomach is dilated.

Gastroptosis is another affection which may cause rather vague symptoms of indigestion. But the surgeon who is acute in eliciting a patient's clinical history will not easily be misled into mistaking gastric dilatation for gastroptosis or *vice versa:* because in the case of obstructive dilatation it is exceedingly rare for the clinical history to be negative; while in uncomplicated cases of gastroptosis it is the rule. In uncomplicated cases of gastroptosis, we say; for it has been our experience that the proptosed stomach is always dilated, unless it forms a part of a general visceroptosis.

Prognosis.—In secondary gastric dilatation the prognosis is bad, unless the mechanical obstruction be relieved by mechanical means. All that was said on the prognosis of gastric ulcer in general, should be borne in mind in this connection. It was there (p. 98) pointed out that under the best medical treatment the death-rate from gastric ulcer in general is at least 10 per cent., with a large proportion of relapses; but that after timely operation, all but from two to five per cent. of the patients recover, and most of them remain permanently cured. The statistics from which these conclusions were drawn included not alone cases of open gastric ulcer, but those cases where the stomach was very extensively diseased—dilated, distorted, or contracted as a result of chronic ulceration. We possess, unfortunately, no series of statistics by which we can compare the results in patients with gastric dilatation who have been treated medically, with those obtained in the same class of patients after operation. The large masses of statistics hitherto published include all stages of gastric ulcer; and it is only because gastric dilatation is a more serious affection than gastric ulcer without dilatation that conclusions which are justly drawn from statistics of the disease in general, apply with greater force to its more serious aspects. But in the case of **gastric tetany,** we may speak in figures with some authority. This affection enjoys a mortality under medical treatment of from 70 to 80 per cent. Although few operations so far have been done for its relief, and though the mortality is severe, yet when compared to the figures just given it is low. Cunningham (1904) collected 8 operations for gastric tetany, with 5 recoveries and 3 deaths, a mortality of 37.5 per cent. To these McKendrick (1907) added 16 successful cases, making a total of 24 operations with only 3 deaths, a mortality of only 12.5 per cent. In the three fatal cases (reported by Fleiner (2 cases), and Gumprecht), death was due to visceral disease, to pneumonia, and to peritonitis.

But it must also be remembered that where a mechanical obstacle exists to the evacuation of the stomach it will be only a question of time until the patient starves to death even under the most energetic medical treatment. The starvation is slow, and it is barely possible that the patient will not recognize the fact that he is starving to death; but the intelligent onlooker, be he physician or layman, appreciates the true seriousness of the patient's condition; and it is no longer necessary for the surgeon to urge that in such cases surgery affords the only escape from death. What the surgeon still urges, is that the operation shall be undertaken while yet there is sufficient

recuperative power left in the body cells of the wretched patient. Perhaps the day will come, but it has not yet dawned, when the surgeon will no longer need to urge even this, but when all physicians will, as at the present time the most progressive of them do, invite the surgeon to see their stomach cases with them, in order that they may decide, in the light of the knowledge the physician can shed on the case, not only whether an operation is required, but also at what period of the disease it had best be undertaken. We have no hesitation whatever in saying that when obstructive dilatation of the stomach is once diagnosed, all delay should be avoided, and surgery should at once remedy the mechanical defect which Nature and her handmaid Medicine are unable to remove.

Treatment.—The choice of operation lies between gastro-jejunostomy, pylorectomy, and pyloroplasty. Unless suspicion of malignancy is entertained, we think pylorectomy for gastric dilatation is to be condemned. As a rule, patients with severe grades of gastric dilatation are not good surgical risks, so that pylorectomy often is unjustifiably severe; for the same reason we should propose excision only in an extremely small number of these cases. We believe that gastro-jejunostomy will continue to give in the future, as it has in the past, the best results; and that Finney's operation should be reserved for those patients in whom gastric motility is but slightly impaired. This would limit its application to gastric dilatation to the earliest stages of the disease. It is certain, moreover, that gastro-jejunostomy gives more immediately gratifying results in patients whose pylorus is almost impassable even to liquids; so that in the earlier stages of dilatation, where the pylorus is still slightly patent, Finney's operation may be preferred.

Gastroptosis

Gastroptosis, a condition in which the whole stomach is displaced downward, sometimes requires surgical treatment. The causes of the affection are obscure. Glénard, in 1885, drew attention to general visceral prolapse involving, besides the stomach, the intestines, usually the right kidney, and sometimes the liver and spleen as well. To account for these changes, various theories, none of them very satisfactory, have been advanced. Only a few etiological factors seem to be susceptible of demonstration. It is a condition which is very much more frequent in females, and good reasons exist why this should be so. Apart from the influence of the clothing of that sex, including the use of corsets, the practice of tight lacing, and of suspending heavy skirts from

the waist instead of from the shoulders or hips, there are the well known influences of repeated pregnancies and childbirths in relaxing the abdominal walls and weakening the pelvic floor. All these mechanical factors tend to allow a descent of the structures in the upper abdomen. Scoliosis, and other deformities of the skeleton which reduce the area

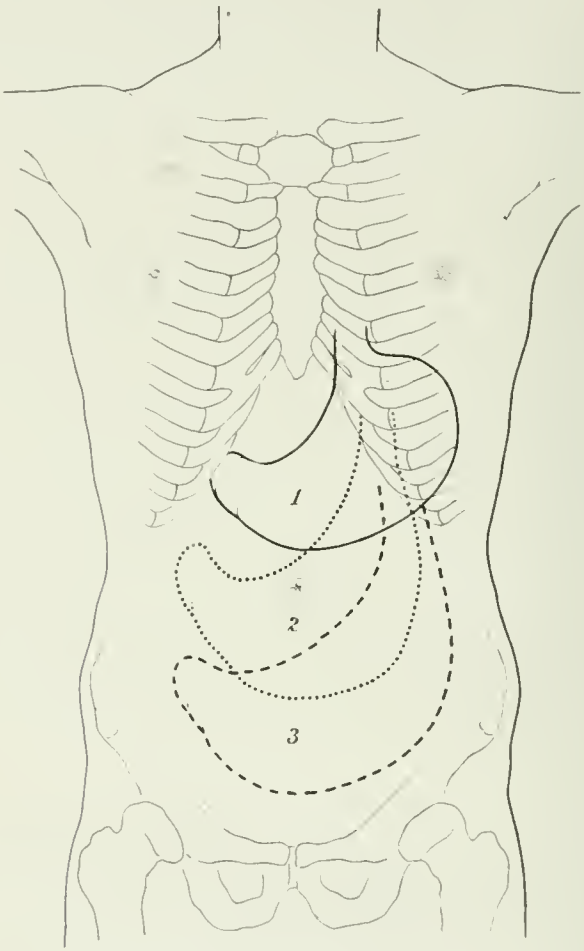

FIG. 43.—Various Degrees of Gastroptosis.

of the upper abdominal regions, are also considered by some to be causes of gastroptosis. Sudden loss of flesh, as in wasting diseases, such as typhoid fever and severe attacks of influenza, is thought, and sometimes with apparent good reason, to be a cause of gastroptosis. The influence which a dilated stomach exerts, both by its weight and its atony, has been too little appreciated; and when once gastroptosis is

added to dilatation, food stagnation is mechanically favored, and one condition continues to aggravate the other. A floating kidney, which by its weight displaces the duodenum and transverse colon, is a well recognized factor in the development of gastroptosis.

The **clinical pathology** of gastroptosis is of some importance. We may recognize three groups (Fig. 43). (1) Where the greater curvature of the stomach is still above the umbilicus; (2) where the lesser curvature is still above, though the greater has descended below the navel; and (3) where even the lesser curvature has passed below the umbilicus. The stomach is usually dilated; among 32 cases of gastroptosis studied by Worden (1906) there were only 3 in which the stomach was not dilated. The gastro-hepatic and gastro-phrenic omenta are stretched, as is the transverse meso-colon; the transverse colon sags and the stomach becomes more or less horizontal, lying in the transverse rather than in the longitudinal axis of the body; and in extreme cases the pylorus itself descends, dragging the first and second portions of the duodenum with it. The transverse duodenum is usually so securely fixed that its position does not change, but sometimes it is found lower than normal, crossing the fourth, or fifth, instead of the third, lumbar vertebra. These changes naturally are prone to cause a kinking of the pylorus, and will add to the gastric dilatation usually present.

Symptoms are occasionally absent in cases of gastroptosis, even when the displacement is well marked. In other patients a very slight degree of gastroptosis causes very distressing and disabling symptoms. Those of neurasthenia are frequently more pronounced than the symptoms referable to the stomach itself. In general the symptoms resemble those of dilated stomach. There is flatulence after eating, occasionally so pronounced as to constitute peristaltic unrest of the stomach. The clothes are loosened and in severe cases the reclining position is habitually assumed after meals. Large meals are avoided; and so painful may the process of digestion become that patients will almost starve themselves rather than endure it. Emaciation is the usual sequel. The pain is a tearing or a stretching sensation, as a rule easily distinguishable from the intense boring pain of gastric ulcer or cancer.

From symptoms alone it is rarely possible to reach an accurate diagnosis. **Physical examination** is much more satisfactory. *Inspection* of the abdomen, with the patient standing, usually reveals a protruding lower abdomen, not due to fat, for these patients are usually emaciated, but to the descent of the stomach from the epigas-

tric to the umbilical or hypogastric regions. The epigastrium is empty and hollow, and frequently the pulsations of the aorta are visible below the ensiform process. On palpation this pulsation can almost always be felt with abnormal distinctness. The contour of the lower chest, showing the effects of corset pressure, is of diagnostic value. Some authors have insisted upon the mobility of the tenth rib, as a predisposing cause, allowing undue pressure upon the liver, and through it displacing the stomach. *Palpation and percussion*, especially when the stomach has been distended with air or fluid, will readily enable the examiner to outline the greater curvature, and in severe cases the lesser curvature also may be detected in this manner. *Skiagraphy* may be employed though we incline to the opinion that a correct diagnosis may almost always be made without it; and it is possible that in some cases the weight of the bismuth or barium meal may cause the appearance of ptosis when none is present.

Treatment.—As it is becoming more and more fully appreciated that gastroptosis is not an isolated condition, but almost invariably a part of a general visceroptosis, it is no longer the custom to limit surgical treatment to the stomach alone. The condition often verges into that broad and rather hazy field of intestinal stasis; but in these pages no attempt will be made to discuss this vexed question. It is merely desired to call renewed attention to the fact that non-operative measures frequently are effectual and always should be tried before opening the abdomen. Especially valuable is forced feeding, the patient being confined to bed for several weeks, with the foot of the bed elevated to overcome the effect of gravity. General and abdominal massage should be given. These measures almost always will cause a deposit of fat in the mesenteries and omenta, which will give sufficient support to the fallen viscera to overcome the distressing symptoms. When sufficient weight has been put on, the patient may be allowed to sit up for a short time each day, and may gradually (very gradually) resume active life. Elevation of the foot of the bed and careful dieting (forced feeding, not abstinence from food) should be continued for many weeks after presumed convalescence. A properly fitting orthopedic corset, which supports the lower abdominal wall while leaving the upper abdomen and lower thorax free, should be worn constantly when out of bed. Such a support should be shown by fluoroscopy to hold the viscera in proper position.

Operative treatment is rarely called for in cases of gastroptosis. Various methods of operating have been adopted. *Gastroplication*, proposed as a remedy for dilated stomach in 1891 by Bircher, was first employed by Summers in 1897 in a case of gastroptosis. It was

adopted also, with success, by Hodge (1906) in combination with Beyea's method of gastropexy to be presently described.

Duret, in 1896, was the first to resort to operation for the relief of gastroptosis. He did a *gastropexy*, suturing the anterior gastric wall to the parietal peritoneum. Rovsing and Hartmann employed similar operations. Beyea in 1899 described a second method of operating by gastropexy which he had practised first in 1897; he shortened the gastro-hepatic omentum by a series of interrupted sutures. About the same time, according to Blecher (1900), Bier devised and employed a similar operation, though an account of his method was not published until later. Coffey (1902) sutured the root of the great omentum to the abdominal wall, thus giving support to the stomach from below. Gastro-jejunostomy has also been employed by a number of surgeons; it was particularly supported by Hammer (1903), who came to the conclusion, both from a review of the literature, and from his own experience, that it was preferable to any other operation. Such was formerly our own opinion; we had found no cases of gastroptosis unaccompanied by gastrectasis, and although the relief of symptoms following gastro-jejunostomy under such circumstances has not been so pronounced or so constant as in patients with dilatation of the stomach without gastroptosis, yet it was satisfactory both to patient and surgeon. It is undeniable, however, that the profession as a whole does not regard gastro-jejunostomy as the best form of operation for gastroptosis. This is so, we believe, because it is becoming more and more recognized that the ptosis of the stomach is not the only, even if the most conspicuous lesion; it is but a part of a general visceroptosis, accompanied by intestinal stasis, which usually requires more extensive operative interference, if the patients' symptoms cannot be relieved by non-operative methods.

The theoretical objections to gastropexy by Duret's method are the interference with the motility of the stomach and the liability of the newly formed adhesions to cause more discomfort than the original disease. But Rovsing, according to Coffey (1912), has reported 163 cases treated by this method, with 92 complete cures (over 56 per cent.), 34 greatly improved, 17 slightly improved, 24 unimproved (under 15 per cent.), and only 8 deaths. From these particular theoretical objections to Duret's method, the *operation of Beyea* is free, since the stomach is raised approximately to its normal position by shortening the gastro-hepatic omentum, without the formation of adhesions to the stomach itself. Beyea stated (1913) that he had

resorted to this operation in 43 patients, 40 of whom were traced: 23 were classed as cured (53.5 per cent.), 14 as improved (32.5 per cent.), and only 3 were not improved.

Omentopexy.—Coffey reported in 1912 the end results in 40 patients with gastro-intestinal stasis (due to causes other than stricture or ulcer) who had been treated by his "hammock operation" of omentopexy. These were mostly cases of enteroptosis. He obtained 26 symptomatic cures (65 per cent.); 9 great improvements (22.5 per cent.); 4 slight improvements (10 per cent.); and only one failure. This patient had tuberculous ulcers of the intestines. We believe, with Coffey, that in many cases a combination of operative measures will bring the best results; Coffey recommends: first, shortening of the natural supports of the liver and stomach by the method of Beyea; second, suture of the great omentum (just below the transverse colon) to the abdominal wall, above the umbilicus, with sutures which pass entirely through the omentum; third, expand the upper abdomen and contract the lower by plastic operations on the abdominal walls. Coffey correctly insists that these methods are to be employed only when non-operative measures have been faithfully tried without relief.

Douglas (1915) traced eight out of ten patients who had been operated on by a combination of Coffey's and Beyea's methods: 7 showed marked improvement, and 1 was not benefited although weight had been gained. X-ray examinations showed the stomachs usually in a lower position than soon after the operations, but in no cases as low as before operation.

In all cases treated by operation, the same careful attention to methods of increasing the deposit of fat in the mesenteries and omenta, and proper support of the abdominal walls, should be adopted, after operation, as advised in speaking of non-operative treatment.

CHAPTER VI

OBSTRUCTION OF THE CARDIAC ORIFICE OF THE STOMACH

Congenital Imperforation of the Esophagus.—Although this affection is extremely rare, and is seen rather by the pediatrist than the general surgeon, yet it seems worthy of short notice in this chapter, inasmuch as gastrostomy or some similar operation presents the only hope of cure.

The subject was well reviewed by Demoulin (1904) and by Zeit (1912), the latter of whom succeeded in collecting fifty-five recorded cases. The gastric portion of the esophagus communicated with the trachea in 44 of 50 cases studied by Demoulin, and with the bronchi in 2 cases. It is therefore extremely unusual for the malformation to consist of a simple obstruction of the lumen of the esophagus by a membrane, or even for the two portions of the esophagus to lie in the same axis, connected by a fibrous band. The symptoms, which exist from the time the child begins to take nourishment, consist (1) in the constant and persistent regurgitation of food, and (2) in the recurrent attacks of smothering which are recognized as characteristic of the disease. These smotherings are due to the regurgitation of mucous and gastric juice into the air passages, through the gastric portion of the esophagus. If the baby does not die of asphyxia in one of these attacks, pneumonia may occur from the regurgitation of gastric fluids, or from the inspiration of food. Inanition will quickly kill the infant should he escape all other perils.

Operative treatment, according to Demoulin, was first suggested in 1866 by Tarnier, who proposed gastrostomy. Steel (1888) was the first to perform gastrostomy. His patient was twenty-four hours old, and died in twenty-four hours. In 1903 Robineau again operated, on the third day of life, by gastrostomy; but his patient died on the third day. Villemin's case, reported by Demoulin, was operated on in 1904, at the age of three days, by gastrostomy; this patient lived five days after the operation. Kirmisson (1904) reported the fourth fatal case, operated on at the age of three days

by gastrostomy. Putnam (1906) has added a fifth fatal case, and Brennerman (1913) two other cases of operation (jejunostomy in one, gastrostomy in the other), both unsuccessful.

The question naturally arises, in view of the extent of the malformation, whether any operation can be expected to be of benefit. Broca has expressed himself as unalterably opposed to any operation on an infant so malformed.

In discussing Demoulin's paper, Broca gave as his opinion that death is the best solution of the difficulty; that he had been gratified to learn that all the patients operated on had died; and that on this account he was glad to study with Demoulin new and more complicated operations, because these will be still more certain to result in death. Never, he said in conclusion, would he assume the responsibility of putting into circulation in the world an infant with its mouth in the duodenum! To our mind, such an infant is no more of a monstrosity than one whose alimentary tract empties into the bladder, or possesses no opening at all at its lower extremity; and we fail to see why, if operation be justifiable in one case, it will not be equally so in the other. The surgeon is not an executioner. It is not for him to decide whether an individual is fit to live or not. His duty is to prolong his patient's life, and to use the agencies of modern surgery in the attempt to overcome deformities and to restore the malformed to a state as nearly normal as possible. It may be objected to this reasoning that a patient who already has a malformation of his esophagus is rendered only more abnormal by the formation of a gastric fistula. Such a reply, we submit, is not argument, it is repartee. But it may be further argued, that even were the patient who has submitted to gastrostomy to survive the perils of infancy—that even were he to reach an age when a more serious operation might justifiably be undertaken—it might be said that even at that period of his life surgery could offer no permanent solution of the difficulty; in other words that the restoration of an esophagus whose upper end is a blind pouch, and whose lower end opens into the trachea, is a problem beyond the possibility of solution by surgery. For our own part, we do not take so narrow a view of the surgical possibilities of the future. We have, on the contrary, the utmost confidence that all problems of mere technique will ultimately be solved. We cannot, of course, hope to make a new esophagus grow; but given the patient, fit for an operation for the restoration of such an esophagus, and we doubt not that some surgeon will solve the problem of the technique. It may not be in Broca's time, nor even during our own lives; but we are none the less confident that such

a time will come.[1] We therefore give it as our unqualified opinion that, save in the already moribund, the surgeon is not only justified in resorting to operation, but he would be worthy of condemnation should he refuse to employ the skill he possesses in the attempt to give these patients a fighting chance for life.

Gastrostomy has heretofore been the only operation employed. On account of the danger of liquids, injected into the stomach by the gastric fistula, entering the lungs by way of the esophageal communication, Demoulin suggested that jejunostomy would be a safer operation. If this could be safely combined with ligation of the pylorus, and gastrostomy as well, a state of affairs temporarily satisfactory might be obtained. If the pylorus were not ligated, bile and pancreatic juice, and possibly also the injected food stuffs, might find their way into the stomach; and unless the stomach, even when excluded from the digestive tract, as by ligation of the pylorus, were drained exteriorly, it would still discharge its secretions into the trachea, and so threaten death from suffocation. Possibly gastro-jejunostomy combined with gastrostomy, by passing a tube through the gastrostomy wound and the gastro-jejunostomy into the jejunum (the method of Rutkowski and Witzel) might accomplish the same result. But any operation on infants a day or so of age must be simple and quick; and for these reasons we prefer gastrostomy. Roux's method of subcutaneous gastro-esophageal anastomosis or one of its modifications (p. 322) will afford the patient a chance for ultimate cure, should immediate death be averted.

Cardiospasm; Diffuse Dilatation of the Esophagus.—This is not an affection of great rarity. Plummer (1912) has had 130 cases under his personal observation.

Its pathogenesis is obscure. It was formerly held that it was strictly analogous to pylorospasm; but this is merely begging the question, as the pathogenesis of the latter is also obscure. Most authorities at present believe that there is some disturbance of innervation of the esophagus or of the cardia; very few cases are due to actual lesion (ulcer, stricture) at the cardia (12 out of Plummer's 130 cases had such lesions). A polyp was the cause in a case reported by

[1] The above paragraphs were written before the publication (January, 1907) of the daring operation by which Roux of Lausanne seeks to form a new esophagus by transplanting beneath the skin of the sternum a coil of the jejunum excluded from the intestinal tract; and also before the appearance of Baudouin's article (1907), where, being ignorant of Roux's operation, he proposed to connect an esophagostomy opening in the neck, with a gastrostomy opening in the epigastrium, by means of a rubber tube, or some similar contrivance. (See page 322.)

Ledderhose (1904). Bassler (1914) believes that the trouble is not in the esophagus or cardia at all, but consists in a spastic contraction of the esophageal opening in the diaphragm, producing obstruction above the cardia.

Symptoms.—Mild cases often pass unperceived. The patient may feel that the food lodges a moment before entering the stomach, and may be able to force it through voluntarily, by taking a long breath and contracting certain of the pharyngeal and esophageal muscles. Such patients as these usually are neurotic or hysterical females; 24 of Plummer's cases were of this type, and in none was there diffuse dilatation of the esophagus. In more severe cases, usually patients without a neurotic taint, a pouch develops; the patient, eating little at a time, will form a habit of retiring to a quiet nook after taking food, and will there wrestle with the obstruction until either it gives way or the distress is relieved by vomiting. In these cases no anatomic stenosis is found; of Plummer's cases, 91 were of this type. Passage of a bougie in such cases usually detects both the obstruction and the pouch. The diagnosis is aided by direct esophagoscopy and by X-ray examinations.

Treatment.—In the hands of an expert the best treatment is divulsion of the obstructing area by means of some form of baloon dilator passed down the esophagus. This was adopted by Russell in 1898, Rosenheim in 1902, and has been extensively employed by Plummer since 1906. Taking the largest class of cases, those patients with diffuse dilatation of the esophagus, without anatomic stenosis, in Plummer's series, 91 in number, it is interesting to learn that he had knowledge of the end results in 84 cases; 3 patients could not be traced, 3 had died of intercurrent affections since operation, and 1 died as a result of the operation, from rupture of the esophagus (pressure of 720 mm. of mercury, but without pain during application of this pressure); 73 patients were found to be completely cured from 1 to 6 years after operation, and 11 were not completely cured, still presenting annoying symptoms, which however, might probably be relieved by further divulsion. Two or three divulsions are given at intervals of a few days; then if no food residue is found in the esophagus after a ten day interval without treatment, the patient is dismissed until further symptoms arise. Plummer considers a pressure of 575 mm. of mercury relatively safe; but absence of pain is not a safe indication of the amount of pressure permissible.

Divulsion of the cardia after gastrotomy has been employed occasionally in these cases for over a generation. The late Prof.

Ashhurst, in 1893, referred to cases of this kind in the hands of Loreta, v. Bergmann, Catani, Frattini, and Billroth. Mikulicz reported six such operations, no recurrence of the trouble being noted in any case. Ledderhose's patient, already referred to, recovered after removal of a polyp from the lower end of the esophagus. Other cases of direct divulsion of the cardia after gastrotomy have been recorded by Erdman (1906), and E. Martin (1906); both patients obtained permanent relief of symptoms. After opening the anterior wall of the stomach the cardia is divulsed digitally (Mikulicz employed forceps with

FIG. 44.—Exposure of Cardia after Left Lateral Ligament of Liver has been Divided and the Liver Drawn to the Right. 1, 1a, Cut edges of Left Lateral ligament; 2, Lower End of Esophagus; 3, Non-peritoneal Surface of Diaphragm; 4, Re-entrant Angle between Liver and Diaphragm; 5, Non-peritoneal Surface of Liver. (*Lambert, in Surgery, Gynecology and Obstetrics.*)

rubber covered blades) until two or even three fingers will readily enter the esophagus. The stomach is then closed, replaced, and the abdominal wound repaired without drainage.

Cardioplasty, analogous to the pyloroplasty of Heineke-Mikulicz, was employed by Wendel (1910). The cardia was exposed by temporary resection of the left costal border; the artery crossing the cardia was doubly ligated and divided; and the esophagus was drawn down into the abdomen far enough to be temporarily ligated with tape above the cardia. The stomach was then clamped, and a longitudinal incision made through the lowest part of the esophagus through the

cardia into the stomach. This incision was then closed transversely; the diaphragm was stitched to the newly formed cardia, and the operative field covered by suturing the stomach around it to the diaphragm. A gastric fistula was formed for temporary feeding. Recovery was prompt and permanent. Lambert (1914) employed a similar operation, obtaining good exposure of the cardiac orifice by detaching the left lateral ligament of the liver (Figs. 44, 45); he introduced (through a gastrostomy, opening) a clamp, to act on the principle of Dupuytren's enterotome, one blade being placed each side of the contracted cardia,

FIG. 45.--Exposure of Cardia and Esophagus. 1, Cut Edge of Left Lateral Ligament of Liver; 2, Dilated Lower End of Esophagus, Drawn Through Diaphragmatic Opening; 3, Which has been Incised; 4, Re-entrant Angle between Liver and Diaphragm. (*Lambert, in Surgery, Gynecology and Obstetrics.*)

and the clamp being gradually tightened until it cut through the walls of the esophageal pouch and the fundus of the stomach. His patient recovered excellent health.

Willy Meyer (1911 and 1912) obtained a cure in three cases of cardiospasm by *plication of the dilated esophagus* by means of thoracotomy. He was inclined to believe, however, that the most valuable part of the procedure was the separation of the vagi from the esophagus, and that the plication may have been unnecessary.

Cicatricial Contraction of the Cardiac Orifice of the Stomach, from other than malignant disease, is so extremely rare that it need only be

mentioned in passing. When, as is less infrequently the case, it is caused by the ingestion of corrosive liquids, the symptoms are overshadowed by those of esophageal stricture, but when this tube is not involved, the usual symptoms of cardiac obstruction are present, but without the cachexia which so early develops in cancer. If bougies (which should be passed only under the control of direct vision, through an esophagoscope) fail to keep the passage open, internal esophagotomy (by direct esophagoscopy) may be attempted; but divulsion, as recommended for cardiospasm, is too unsafe for cicatricial contraction. Or gastrostomy may be done, and the cardia may be cautiously dilated instrumentally or by the fingers, and retrograde passage of a bougie attempted. Much good may ensue, as in stricture of the esophagus, from attaching a string to the bougie, and drawing it out of the mouth. The stricture may then be sawed by means of the string, whose two ends, passing from the mouth and the gastrostomy wound, may be tied together and thus kept safely in place. Or gradually increasing sizes of rubber tubing may be drawn through the stricture by means of the string. It is well in any case to keep the gastric fistula patulous for a number of months; it may be used from time to time while some passing irritation of the cardia is subsiding, and until nourishment may be taken again in the usual way.

CHAPTER VII

HOUR-GLASS STOMACH AND GASTRIC DIVERTICULA

Hour-glass Stomach.—This term well describes the condition found in the immense majority of patients in whom the stomach is loculated; but as cases are occasionally observed in which three (Moynihan, Paterson, Kausch, Schmitt) and even five pouches (Klein) exist, the term segmented stomach, advocated by Wölfler (1895), is more generally applicable. And as diverticula of the stomach are produced by essentially the same causes as those operative in cases of hour-glass contraction, it is convenient to consider them both in the same chapter.

The condition, first noted by Amyand (1734), was subsequently described by Morgagni (1761). The earlier writers on the subject, and those even until recent years, considered it a congenital anomaly in the vast majority of cases. Later writers, notably Moynihan, have proved that as a congenital deformity it is of the utmost rarity, if indeed not altogether unknown. Moynihan is not willing to accept as genuine examples of congenital deformity any of the cases whose records he has examined, nor has his study of museum specimens altered his opinion. Delamare and Dieulafé recorded in 1906 the case of a bilocular stomach in a new born baby, born of syphilitic parents, but with no syphilitic lesions itself; in this case the only lesion found, even on microscopical examination, consisted in hypertrophy of the muscular coat at the junction of the cardiac and the pre-pyloric portions. Gardiner (1907) observed hour-glass contraction of the stomach, associated with an *accessory pancreas*, at autopsy on a child three months old. It must further be remarked that as recent anatomo-physiological researches have called renewed attention to the stomach, we realize the truthfulness, heretofore almost forgotten, of the descriptions of normal stomachs long ago made by Home, Cruveilhier, Henle, and others; and we are thus able to explain as normal many appearances found post-mortem which were at one time considered pathological (see Chap. II, p. 46). Moreover, even in cases of hour-glass stomach observed in infants and young children, it may be quite possible for the deformity to be explained as due to pre-existent disease of the stomach in infantile or intrauterine life.

While, therefore, it cannot be categorically denied that such a thing as a congenital hour-glass stomach may occur, it must be acknowledged to be of extreme rarity; and any cases reported as such deserve prolonged and critical investigation.

Schomerus (1904) found that among 1014 operations for gastric lesions, 71 or 7 per cent. were for hour-glass stomach. Among 154 operations for hour-glass stomach which he studied, 128 were in females and 26 in males. The chief **cause** of acquired hour-glass contraction is preceding *gastric ulcer*, but some cases are due to *cancer*, usually to that form which has developed as a consequence of benign ulceration; others are caused by pressure of neighboring organs, as corset liver Rasmussen (1887), according to Schomerus, thought that hour-glass stomach might be caused by *pressure of the left costal border*; some cases are caused by *perigastric adhesions*; and a few are produced by the *ingestion of corrosive liquids*, as in a case recorded by Klein, in which operation was done by Schnitzler (1900). Other cases of hour-glass stomach, due to the ingestion of acids, have been recorded by Carle, Gersuny, Hacker, and Korte. *Syphilitic ulceration* is a rare cause (Guillemot, 1899). Langenbuch recorded a case accompanied by *tuberculous ulceration* in both pouches; but the etiological relation of the ulcers was doubtful.

Clinical Pathology.—The constriction is usually single, situated somewhat nearer the pyloric than the cardiac orifice, and the greater curvature is more often drawn up toward the lesser, than the reverse. But while these are the usual characteristics, a great variety of deformities has been encountered. In the cases studied by Schomerus the constriction was near the pylorus in 51, midway between the orifices in 34, and near the cardia in only 13 patients.

When the pyloric pouch is large there are two dangers—the first and more common is that at operation the cardiac pouch may be entirely overlooked, and a gastro-enterostomy done with the pyloric portion, without improving the patient's condition. This error, according to Lieblein and Hilgenreiner (1905), has been made by Bier, Czerny, Kuster, Hartmann, and others. All the known cases have been attended by a fatal result. The other danger is that an unusually large pyloric pouch may be the seat of *volvulus*, as in cases recorded by Langerhans, Doyen, and others, the greater curvature ascending toward the left, and adding the factor of strangulation to the pre-existent obstruction. Volvulus of a large cardiac pouch does not appear to have been observed.

The frequency with which pyloric stenosis complicates hour-

glass stomach has been much emphasized by Robson and Moynihan. The stenosis in both situations may be due to ulcer, or one may be caused by perigastric adhesions These adhesions may act as a bridle, passing across the stomach from one curvature to the other, or the stomach may itself become adherent to the neighboring organs or to the anterior abdominal wall. In one case of trifid stomach, recorded by Robson and Moynihan, both constrictions were due to ulceration; in their second patient one constriction was caused by ulcer, the other by adhesions. Dilatation of the duodenum should not be mistaken for hour-glass stomach. Christian (1907) has recorded an interesting case in which such an error was made.

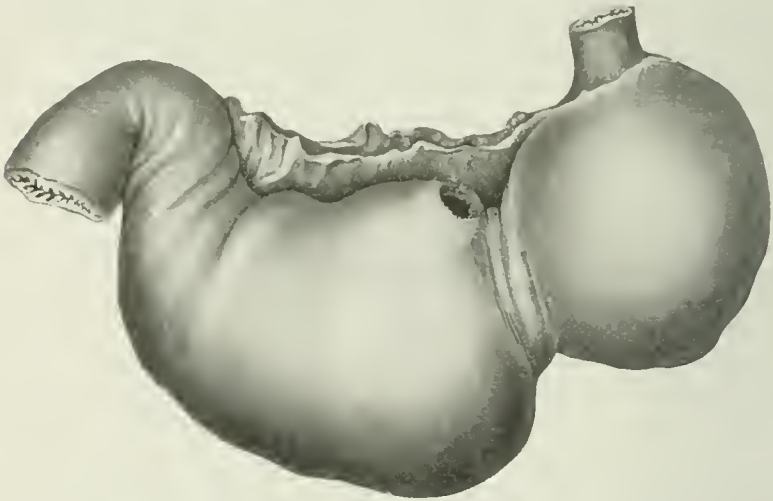

FIG. 46.—Hour-glass Stomach from Carcinomatous "Saddle" Ulcer on Lesser Curvature with Perforation. From a Patient in the Episcopal Hospital. (*Half Natural Size.*)

A saddle ulcer on the lesser curvature is a frequent cause of hour-glass stomach. In a case reported by the junior author (1902) this ulcer showed beginning carcinomatous change (Fig. 46). The condition in one patient at the Lankenau Hospital where the contraction was formed by extensive perigastric adhesions is shown in Fig. 47.

In the majority of patients under our care, however, the constriction has been due to cicatrization of benign ulcers. In one patient the lesion was thought at operation to be malignant; but as the patient remained in good health more than four years after the operation (gastro-gastrostomy and Finney's pyloroplasty) it is evident that the clinical diagnosis was erroneous.

The **symptoms** of hour-glass stomach are rarely distinguishable from those due to pyloric obstruction caused by ulcer. If the constriction

is close to the cardia, the clinical picture simulates obstruction of this orifice. In most of the recorded cases the condition has been found at autopsy, or has been met with unexpectedly at an operation for the relief of long-standing gastric symptoms usually thought to have been caused by ulceration at the pylorus.

Hour-glass constriction is one of the latest results of gastric ulceration. Frequently no history of acute ulceration can be discovered; and it is almost always certain that the condition when met with at operation has existed for many years.

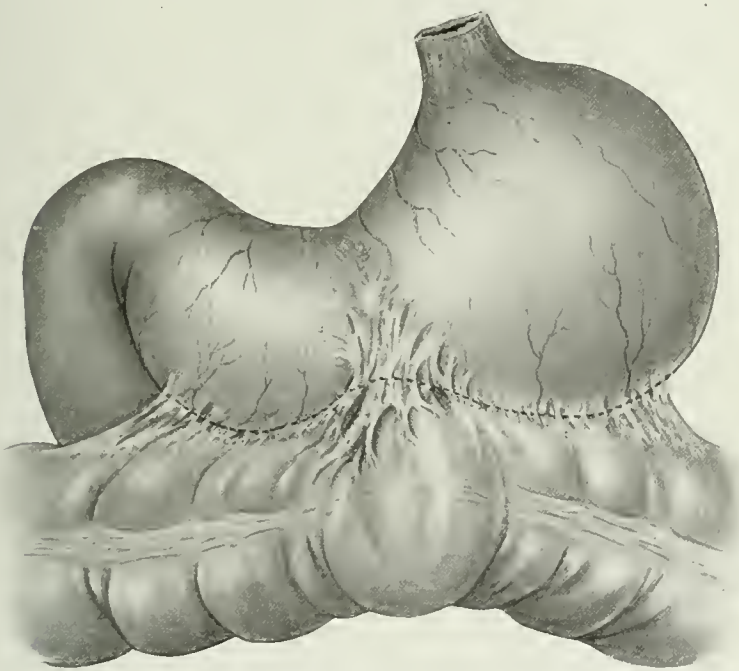

FIG. 47.—Hour-glass Stomach from Perigastric Adhesions (Gastric Ulcer). From a Patient in the Lankenau Hospital. (*Half Natural Size.*)

By **physical examination** it is sometimes possible to make a positive diagnosis before opening the abdomen. Moynihan in his first six cases made a correct diagnosis only once before operation; among his next nine patients, however, he made the diagnosis in seven with reasonable certainty before operation. The detection of hour-glass contraction by physical examination depends largely upon the use of the stomach tube or skiagraphy. On filling the stomach with liquid through the tube, v. Eiselsberg noticed that a prominence appeared first in the left hypochondrium, and that a few seconds later this swelling subsided,

and a second, further to the right, made its appearance. The passage of fluid from one compartment to the other may sometimes be detected as a gurgling sound. The stethoscope is useful for this purpose, but care should be taken not to mistake the normal deglutition sounds or the pyloric sound for the gurgle due to the passage of liquid through an abnormal constriction. Moynihan, after outlining the empty stomach, gives a Seidlitz powder in two portions; the upper pouch will become distended with carbon dioxide some seconds before the lower. In thin persons the cautious distention of the stomach with air by means of a hand-bulb may render the bi-loculated stomach appreciable to percussion and palpation, or even to inspection. We have already (p. 55) expressed our preference for this method over distention by means of a Seidlitz powder. When, after a measured quantity of liquid has been poured into the stomach, a large portion of it cannot be recovered, it may be assumed that the lost portion has passed into the pyloric pouch (Wölfler's first sign). When, during lavage, the water has all returned clear, and there then comes a gush of cloudy fluid mixed with gastric contents, it has been assumed by Wölfler (the test is known as his second sign) that the clear fluid comes from the cardiac and the cloudy from the pyloric pouch. Under similar circumstances, if it be impossible to recover liquid from the stomach even when splashing may be detected in it, it may be assumed that the fluid and air are contained in the pyloric pouch, where the stomach tube cannot reach. This, which is spoken of as "paradoxical dilatation," is known as Jaworski's sign.

If all these signs were present in any one case it might be safe to conclude that hour-glass constriction of the stomach existed; but as each one may exceptionally be observed in other conditions, notably in marked gastric dilatation, the assertion before operation in these cases that hour-glass stomach exists is in many instances a happy guess.

A more satisfactory outline of the stomach may be obtained by means of fluroscopy, after the ingestion of an opaque meal (see p. 61). Only when the loculation of the stomach remains constantly unaltered during a prolonged period of observation, is it safe to conclude that a true hour-glass stomach is present, since in most cases a pseudo-loculation of the stomach is present due to normal peristaltic contractions, or to temporary spasm caused by an ulceration along the lesser curvature. Downes and LeWald, as noted at p. 225, consider a dumb-bell shaped hour-glass stomach rather characteristic of syphilitic disease of the organ.

Treatment.—When surgical treatment is undertaken for this condition a choice of operation has to be made among the following: (1) Digital divulsion of the constricted orifice by means of gastrotomy; (2) Gastroplasty or Gastro-anastomosis; (3) Gastro-gastrostomy; (4) Gastro-jejunostomy; and (5) Partial Gastrectomy.

The statistics of these various procedures have been investigated by Schomerus. Digital *divulsion* alone seems to have been employed in only one reported case, by Moynihan (1904), who adopted this method under the impression that he was dealing with an inoperable malignant growth. The patient made a satisfactory recovery, was relieved of her gastric symptoms, the tumor disappeared, and she was still in good health more than two years later. Blake (1903) employed divulsion in a patient whose stomach presented a tight stricture close to the cardiac orifice, but as he also did a gastro-jejunostomy, in the pyloric pouch, some of the benefit derived from the intervention may have been due to the latter procedure. In spite of the successful termination in these cases, divulsion is not now an accepted form of treatment, except in rare instances where the constriction is so near the cardia as to be inaccessible from without the stomach. As in the case of pyloric obstruction, divulsion may be regarded as dangerous, uncertain, and in every way less satisfactory than the other forms of treatment to be described.

Gastroplasty,[1] analogous to pyloroplasty, is said to have been performed first by Bardeleben in 1889; this operation was attended by a fatal result; but Krukenberg in 1892 employed it successfully, as did Doyen and other surgeons soon afterwards. Gastroplasty is limited in its application to stomachs where the constriction is benign, unattended by induration or active ulceration, and where the pylorus is not itelf strictured. The employment of pyloroplasty as well as gastroplasty would only be complicating one not very satisfactory operation by another still less promising. Schloffer in one case complicated by pyloric stenosis successfully combined gastroplasty with anterior antecolic gastro-jejunostomy in the pyloric pouch. A modification of gastroplasty, analogous to Finney's pyloroplasty, was introduced by Kammerer (1903), and has been successfully employed by him and

[1] Under the name of *gastroplasty* Nicoladoni suggested the substitution of the transverse colon for the stomach after a circular resection of the latter. Although commended by Kocher, it does not appear to have been employed, and is mentioned here only to avoid confusion of terms. The same term, gastroplasty, is used by Jedlicka to describe an operation consisting in resection of gastric ulcers, with restoration of the gastric wall by the sliding flap method commonly adopted in plastic surgery.

by other surgeons. It may be conveniently designated Gastro-anastomosis. Büdinger (1901) employed, unsuccessfully, a flap method of gastroplasty.

Each method may no doubt be suitable in certain cases, but as a general rule gastroplasty is less successful than one of the methods presently to be described. Schomerus (1904) collected 47 cases of gastroplasty for hour-glass stomach, with 5 deaths (10.6 per cent. mortality); and 5 cases in which pyloroplasty was also done (20 per cent. mortality); as well as 4 cases in which gastro-jejunostomy was performed, with a mortality of 25 per cent. Although the operative mortality (10.6 per cent.) of simple gastroplasty is thus seen to be moderate (some of the deaths cannot be attributed to the operation), the remote results have been disappointing. Paterson found (1906) that "in at least 25 per cent. of the patients who have recovered, either no relief has followed,or relapse has occurred subsequently."

Gastro-gastrostomy, which, under the name of gastro-anastomosis, was first employed, and successfully, by Wölfler in 1894, is even more limited in its application than is gastroplasty. Unless the two pouches of the stomach can be approximated without tension, the operation is not only difficult of execution, but may be attended by a fatal result from giving way of the sutures. It is therefore contra-indicated when the cardiac pouch is small, when the scar is wide-spreading, or when many adhesions are present. In inoperable cases of malignant disease it is possible that some temporary relief of symptoms might be thus obtained, but usually in these, as in benign affections, better results will follow gastro-jejunostomy. The main indication, we think, for gastro-gastrostomy is in the treatment of an hour-glass constriction with large pyloric pouch in the presence of pyloric obstruction, when the pyloric pouch may be successfully drained by gastro-jejunostomy. If pyloric stenosis does not exist, gastro-jejunostomy in the cardiac pouch is to be preferred; though if the symptoms are due to dilatation of the cardiac pouch without marked stenosis of the lumen between this and the pyloric pouch, gastro-gastrostomy may prove effectual, as in Wölfler's patient. We have found references to 44 operations by gastro-gastrostomy, with 5 deaths, a mortality of 11.4 per cent. The end results in most cases are not known, but definite recurrence was noted in 2 patients, 5 and 7 years respectively after operation. A patient aged 79 years operated on by the junior author at the Walter Reed General Hospital for carcinomatous hour-glass stomach, regained his health and resumed his normal life for about 6 months, but then died rapidly from extension of the disease.

Gastro-jejunostomy.—Veyrasset (1908) collected 73 cases in which gastro-jejunostomy had been done for hour-glass stomach. Among these patients, 14 died (19.1 per cent. mortality). Recurrences are extremely rare. Paterson (1906) found only 2 recurrences among more than 30 patients whom he traced; and in one of these the return of symptoms was "clearly due to the coexistence of pyloric stenosis." The anatomosis should of course be made with the cardiac pouch; and as the existence of this pouch, and consequently the presence of hour-glass stomach, has been overlooked at operation by some very competent surgeons, it is well to bear in mind Moynihan's advice, always to make a point of examining the whole stomach from esophagus to duodenum, before undertaking any operation on it whatever. If pyloric stenosis coexists with hour-glass constriction, simple gastro-jejunostomy will not effect a cure, unless the pyloric pouch be very small indeed.

Hacker was the first (1895) to consider the treatment of *double gastric stenosis*, and the principles which he then laid down guide the surgeon still. His proposals were: (1) To combine gastroplasty, resection, or gastro-gastrostomy with pyloroplasty, pylorectomy, or gastro-jejunostomy in the pyloric pouch; or (2) that gastro-gastrostomy should be combined in one opening with gastro-jejunostomy, so that, in other words, both gastric pouches should drain through the one gastro-intestinal anastomosis. Mikulicz is said to have adopted this method in connection with a gastroplasty. Finally, v. Hacker proposed a double gastro-jejunostomy, uniting each gastric pouch separately with a loop of the jejunum. This method was also advocated (1896) by Wier and Foote, by whose names it is generally known in this country. According to Monprofit (1904), Clément, of Fribourg, did an anterior gastro-jejunostomy "in-Y" with double lateral anastomosis to the gastric pouches (Fig. 48). Monprofit proposed a double gastro-jejunostomy "in-Y," after Roux's method by implantation; which appears to us a more complicated operation and one no more likely to be successful than that employed by Clément. In

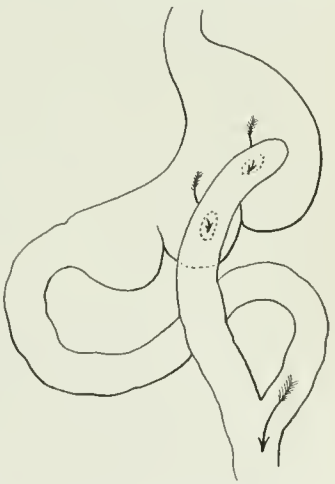

FIG. 48.—Diagram of Double Anterior Gastro-jejunostomy in-Y according to the Method of Clement of Fribourg for a case of Trifid Stomach.

the patient with *trifid stomach* operated on by Moynihan, gastro-gastrostomy was employed to unite the cardiac and median pouches, the constriction between the latter and the pyloric pouch was dilated by the fingers, and the pyloric pouch was drained by gastro-jejunostomy. In Paterson's similar case, gastroplasty was employed to connect the pouches, and gastro-jejunostomy was done in the pyloric pouch, which was the largest of the three. His patient was in good health two years later.

Of these various operative combinations, it appears to us that these are to be preferred: (1) With small cardiac pouch, gastro-gastrostomy or gastroplasty with gastro-jejunostomy in the pyloric pouch; (2) with large cardiac pouch, Finney's pyloroplasty with gastro-jejunostomy in the cardiac pouch; (3) with very small pyloric pouch either (*a*) gastro-jejunostomy in the cardiac pouch alone, (*b*) gastro-jejunostomy in the cardiac pouch combined with gastro-gastrostomy or gastroplasty, or (*c*) lateral gastro-duodenostomy, that is, an anastomosis between the cardiac pouch and the duodenum, as successfully practised in one such case by Schnitzler.

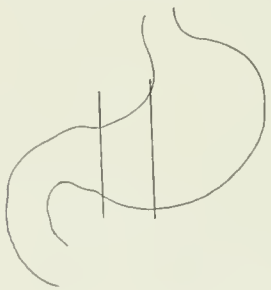

FIG. 49.—Cylindrical Gastrectomy or Sleeve Resection.

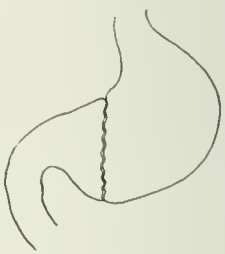

FIG. 50.—Circular Gastrorrhaphy (End to End Suture) after Cylindrical Gastrectomy.

In six cases of hour-glass stomach under the care of the senior author no deaths have occured: gastro-jejunostomy alone was employed in one patient; gastro-jejunostomy combined with gastro-gastrostomy in two patients; while the sixth patient, as already mentioned, remained well more than four years after the performance of gastro-gastrostomy and Finney's pyloroplasty. If there were malignant disease, our preference would naturally be for excision, where practicable. C. H. Mayo (1920) reports from the Mayo Clinic 89 operations for hour-glass stomach, with 7.4 per cent. mortality. He prefers cylindrical resection of the stomach (Figs. 49 and 50), combined with gastro-jejunostomy; and for recurrence advocates partial gastrectomy.

Gastrectomy for hour-glass stomach, even when the obstruction is benign, has been more frequently employed of late years than was formerly the case when it appeared to be unnecessarily severe. Schomerus (1904) collected 8 such operations, with one death. In six cases (Bergmann, Hahn, Körte, Krause, Kümmell, Zeller) circular gastrorrhaphy was done after resection, the lumen of the stomach thus being restored without resort to gastro-jejunostomy. In Hedlund's patient the cardiac pouch was closed by sutures, and then the pylorus was united with the posterior wall of the cardiac portion of the stomach by Kocher's method of implantation. Büdinger, after closing each portion of the stomach separately, did a posterior retrocolic gastro-jejunostomy. The only fatal result was in Zeller's patient (1893), and it was due to the perforation of another ulcer.

Operations for Hour-glass Stomach
(After Schomerus, 1904)

Operation	Cases	Died	Mortality per cent.
Resection	8	1	12.5
Digital divulsion	1	0	00.0
Gastroplasty	47	5	10.6
Gastroplasty and pyloroplasty	5	1	20.0
Gastroplasty and gastro-jejunostomy	4	1	25.0
Gastro-gastrostomy	19	3	16.0
Gastro-gastrostomy and gastro-jejunostomy	2	0	00.0
Gastro-jejunostomy	52	6	11.5
	138	17	12.3

Operations for Hour-glass Stomach
(Statistics published since 1908)

Operation	Cases	Died	Mortality per cent.
Partial gastrectomy	83	6	7.2
Cylindrical resection (mediogastric)	8	1	12.5
Resection (not specified)	12	2	16.66
Gastroplasty	2	1	50.00
Gastroplasty and gastro-jejunostomy	5	0	00.0
Gastroplasty and Finney's pyloroplasty	3	0	00.0
Gastro-gastrostomy	31	3	9.7
Gastro-gastrostomy and gastro-jejunostomy	4	0	00.0
Gastro-jejunostomy	75	7	9.3
Gastro-jejunostomy (double)	5	0	00.0
	228	20	8.7

Among 83 cases of gastrectomy for hour-glass stomach, which we have collected since the publication of the first edition of this work, there have been only 6 deaths, a mortality of 7.2 per cent. In selected cases, therefore, excision appears to be the method of choice.

Gastric Diverticula.—Distinct diverticula of the stomach are very rare. They are divided by systematic writers into those due to pressure, and those due to traction. Every case of dilated stomach from stenosis is really an example of a pressure diverticulum, the deformity in cases of hour-glass stomach sometimes assuming a form more characteristically pouched. Of the distinct diverticula, the form due to traction is less unusual, and is produced by adhesions between the stomach and neighboring structures, especially the pancreas, the liver, and the diaphragm in the region of the cardia. Zahn (1899) observed a gastric diverticulum in which both pressure and traction were probably causative factors. Horrocks (1907) recorded a case which he regarded as *congenital*.

Almost invariably the primary *cause* is gastric ulcer, though a few examples are recorded from carcinoma. If the pouch is so situated and of such conformation as to favor the lodgment of food, it may when thus distended simulate a malignant tumor. Diagnosis before operation or autopsy is almost impossible without the aid of the X-rays. Treatment must be adapted to suit the condition as found. Lieblein and Hilgenreiner (1905) say that in case the diverticulum is small, and not prone to collect gastric contents, a gastro-enterostomy will suffice to effect a cure, by relieving pressure and allowing the distended pouch to contract. Of course gastrolysis must in most cases be an integral part of the operation and it might then become possible simply to invert the pouch into the stomach and close its base by sutures, as in the analogous conditions in the esophagus. In other cases excision of the pouch will be required. This may prove a difficult and dangerous operation. Gastro-gastrostomy might in some cases be preferable.

Few patients appear to have been subjected to *operation*. Kolaczek (1896), in a patient in whom there was present a mass in the epigastric region, adherent to the abdominal wall, made a diagnosis of ulcerating leiomyoma of the stomach. This diagnosis was based on the long duration of the tumor, which excluded malignancy; and on the fact that he considered leiomyoma the least rare form of benign tumor. He excised the mass, which proved to be a diverticulum of the stomach involving the pancreas. The patient recovered and was

reported well six months later. Another operation, by Mosetig-Moorhof, was reported by Silbermark (1904). The diagnosis in this case was osteomyelitis or malignant growth of the left costal margin. Extirpation was undertaken, and the gastric pouch was unwittingly opened, without, however, invading the general peritoneal cavity, which was shut off by adhesions. The tract was found to be lined with mucous membrane, and a sound passed into the stomach through the nose came out through the abdominal wound. A tube was passed into the duodenal end of the stomach from the wound, and the gastric opening was tamponaded. One week later the abdomen was opened in the median line, the stomach was dissected free from the anterior abdominal wall, the edges of the ulcerated area, in which the diverticulum had formed, were freshened, and the stomach was closed by sutures, reinforced by an omental graft. The patient recovered, and was reported in good health one month later. Other operations, in some of which the diverticulum was not recognized until after excision of the diseased portion of the stomach, have been recorded by Barjon and Delore (1912), Chutro (1910), C. H. Mayo (1912), Little (1910), Jones (1909) and Borzĕsky (1914).

CHAPTER VIII

BENIGN DISEASES OF THE DUODENUM

Duodenal Ulcer

Duodenal ulcer, according to Moynihan, was first recognized as a clinical entity by Travers, who in 1817 reported three cases of perforation. Surgical treatment for duodenal ulcer was at first confined solely to the attempted repair of perforations. It was Sidney Jones, in 1888, who first operated on a patient with a duodenal perforation. No diagnosis other than peritonitis was made, and the perforation was discovered only at postmortem examination. Four similar operations, with fatal results, followed this first resort to surgery, and it remained for Gould (1893) to find and to suture the ulcer, although his patient survived only six hours. Five other fatal operations followed Gould's; but finally a patient operated on by H. P. Dean, in 1894, recovered from the operation, but died two months later from intestinal obstruction. The patient of Landerer and Glucksmann (1896) survived six months, and then died of another perforation of the duodenum; but Dunn's patient, operated on in this same year, is credited with permanent recovery.

Fig. 51.—Duodenal Ulcer—Acute, Showing Appearance of Stippling when Serous coat is Abraded by Gauze. From a Patient in the Lankenau Hospital.

Until the publication in 1893 by Perry and Shaw of their studies of duodenal ulcer, the condition possessed little or no surgical interest. Codivilla operated in the same year for symptoms of pyloric obstruction; he found the latter present and that it was caused by a duodenal ulcer. It was Robert F. Weir, however, who first (in 1900) roused surgical attention to the frequency of duodenal ulcer and especially to cases of perforation. No one has done more than Moynihan himself to arouse interest in chronic duodenal ulcer as a disease susceptible of cure by surgical means.

All ulcers on the duodenal side of the pyloric vein, as described by Mayo and by Moynihan, are classed as duodenal. Mayo says: "A

short stumpy vein comes out from above, and another from below, the pylorus." These veins, he points out, are quite unlike vessels in other situations of the stomach, and when once recognized serve readily to locate the pyloric ring. Hartmann, however, who is a keen observer, denies the constancy of the pyloric vein; for our own part, though we have found it recognizable in the vast majority of instances, unless obscured by adhesions, we do not regard it as an infallible indication of the location of the pylorus. Gastric ulcers, except the acute perforating variety, are not very usual close to the pyloric ring; and when a callous ulcer does occur here it is not apt to be mistaken for a duodenal

FIG. 52.—Microphotograph of a fully Developed Duodenal Ulcer. From a Patient in the Lankenau Hospital. (*Dr. Reiman.*)

ulcer so much as for a gastric (pyloric) carcinoma, owing to the muscular hypertrophy and edema which accompany it and give it its resemblance to a tumor. Until these pyloric veins were adopted as a limiting landmark, most duodenal ulcers were classed as pyloric, i.e., gastric, and duodenal ulcer was considered a much rarer affection than gastric ulcer. At present, quite the reverse is true, and in the statistics of all large clinics duodenal ulcers predominate. During a period of seven years at the Lankenau Hospital there were subjected to operation 215 patients with duodenal ulcer, and only 53 with gastric ulcer, or less than one-fifth of the whole series of patients with gastric and duodenal ulcer (Fig. 27, p. 71).

About 95 per cent. of duodenal ulcers lie within 4 cm. of the pylorus and most often upon the anterior or upper surface of the bowel. A certain number of these ulcers actually involve the pyloric ring and may even extend into the stomach. An important variety which often is overlooked, and to which Codman (1909) and more recently Geoffrey Jefferson (1916) have called attention, is an ulcer lying in the duodeno-pyloric fornix (Fig. 53).

In regard to the **causes** of duodenal ulcer, no more is known than of those of ulcer of the stomach. Its usual location lends support to the theory that trauma by the acid chyme as it is squirted through the pylorus may act as a predisposing cause. Mayo (1915) contends that the ingestion of very hot liquids is a predisposing cause, since all liquids are ejected rapidly from the stomach through the mechanism of the *canalis gastricus*, to which reference was made at p. 46. The influence of extensive burns of the body in causing acute ulcerations of the duodenum, while recognized since the days of Curling (1842), has never been thoroughly explained. Alexander (1912) observed this complication in four out of twenty-seven patients with extensive burns.

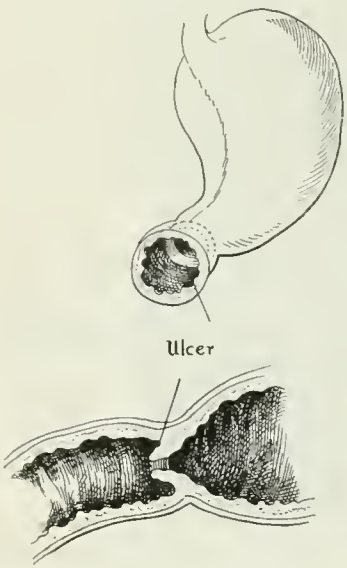

FIG. 53.—Duodeno-pyloric Ulcer. (*After Geoffrey Jefferson.*)

Duodenal ulcer seems to have a predilection for male adults, especially for those between thirty and fifty years of age, whereas gastric ulcer is most characteristic of young women under thirty years of age. Notable exceptions to this rule, however, are occasionally encountered. Genrich, according to Gandy (1899), has recorded a perforation of a duodenal ulcer in an infant 21 hours old; and Torday (1906) reported a duodenal ulcer found at autopsy in an infant of nine months. Hahn, according to Lieblein and Hilgenreiner (1905), found at autopsy on an infant two days old a duodenal ulcer which had caused death from hemorrhage. Cases such as these, in infants, are no doubt of toxemic origin. Among Collin's 273 patients, 16 (6.22 per cent.) were less than one year old.

Symptoms.—While in general characteristics the symptoms of ulcer of the duodenum bear a resemblance to those of gastric ulcer, there

are certain factors sufficiently distinctive, if they be present, to render possible a positive diagnosis of the site of the lesion.

Duodenal ulcer is even more apt to be *latent* than is ulcer of the stomach. Perforation is not unusually the first symptom calling for medical advice. Among 151 cases analyzed by Perry and Shaw the first symptom in no less than 91 was the hemorrhage or perforation from which the patient died. F. Brunner found a history of previous symptoms of duodenal ulcer could be obtained in only 56 per cent. of the patients with perforation whose cases he analyzed. Weir, however, found the previous history positive on this point in 25 (73 per cent.) of 34 cases of duodenal perforation. This corresponds closely with the statistics of the Lankenau Hospital, where a previous history of gastric disease was obtained in 75 per cent. of cases.

Pain in duodenal ulcer typically does not occur until two or three hours after the ingestion of food. Indeed many patients will entirely deny the existence of pain, and only on close questioning will admit that they feel more comfortable when the stomach has some food in it, than they do soon before taking their meals. They frequently acknowledge that they are averse to letting their stomachs remain empty more than two or three hours at a time. They will not sleep soundly the night through unless they have eaten a little late supper just before retiring; or they will be in the habit of taking a cracker and a glass of milk during the night when they wake up in the small hours. All these are manifestations of what Moynihan has graphically called the "hunger pain" of duodenal ulcer. But it is to be noted that patients are rarely aware that it is the discomfort which impels them to keep their stomach constantly occupied. They rather think that the eating at shorter intervals than other people is an idiosyncrasy not based on any pathological cause. It is supposed that the reason the presence of food in the stomach keeps in abeyance the pain, is because thus the acid gastric secretion is neutralized before passing the pylorus, and thus irritation of the ulcer is prevented. Pain is less often *referred* than in ulcer of the stomach.

Vomiting is unusual in duodenal ulcer, unless stenosis of the pylorus is present. *Hematemesis* is equally rare. *Melena*, is not as usual in ulcer of the duodenum as is hematemesis in gastric ulcer. The quantity of blood in the bowel movements, however, is frequently very small, and often escapes the attention of the patient altogether. The tests for *occult blood* are considered by some of great aid in reaching a diagnosis.

Tenderness in duodenal ulcer is almost invariably close to the right costal border, and characteristically is not confined to one spot, but extends through the descending portion of the duodenum.

Jaundice is a rare sign, and is generally thought to indicate that the papilla of Vater is invaded by the ulcer, though it may be due merely to concurrent catarrhal duodenitis, or to independent disease of the biliary tract. We have seen several cases of duodenal ulcer with jaundice from stone in the common duct.

Perforation is usually said to be more frequent than in gastric ulcer. The older statistics, as given by Laspeyres (1902), tended to support this contention: Chvostek found it to occur in 42 per cent. of patients, Collin in 69 per cent., and Oppenheimer in 48 per cent. of patients. But Robson noted perforation only in 10 per cent. of his operative cases; and among 272 operations for duodenal ulcer Mayo recorded only 66 (24 per cent.) for perforation (16 acute, 13 subacute, and 37 chronic perforations). The statistics from the Lankenau Hospital are in accord with Mayo's and with Robson's: in a series of 179 cases of duodenal ulcer, 26 perforations were encountered (23 acute, 3 chronic), or 14.5 per cent.; while in a series of 44 cases of gastric ulcer there were 9 perforations (7 acute, 2 subacute) or 20.4 per cent. In the 59 cases of gastric and duodenal perforation reported by Scully (1918), the 48 gastric perforations formed 9 per cent. of the cases of gastric ulcer seen, and the 11 duodenal perforations formed 15 per cent. of cases of duodenal ulcer seen. It seems probable that the much smaller proportion of perforations in duodenal ulcer in these statistics than in those quoted from Chvostek, Collin, and Oppenheim, may be due to the much more frequent recognition of the symptoms of uncomplicated duodenal ulcer at the present time.

F. Brunner found perforation of duodenal ulcer occurred ten times in the male to once in the female; whereas perforation of gastric ulcer occurred only once in the male to four times in the female.

The perforation is much more frequent in the first portion of the duodenum, and usually is on its anterior wall. Obviously the reason for this is the rarity with which an ulcer is found in other parts of the duodenum. The rarity of perforation of the lower wall, as well as on other retroperitoneal portions of the duodenum, is no doubt due to the protection afforded by the pancreas and other retro-peritoneal structures. Perforations into neighboring organs seldom have been observed. Perforation of the duodenum into the stomach appears to be unknown; that of the stomach into the duodenum, though extremely

rare, has nevertheless been recorded in a few instances. Subphrenic abscess was caused by duodenal perforation in 6 out of 58 cases of the former condition collected by Nowak. Petren (1915) has published notes of six cases, including two personal observations, which he considers undoubted examples of retroperitoneal perforation of duodenal ulcers. All but one of these patients died, though Perry and Shaw's patient lived for more than 5 months after the retroperitoneal abscesses had been incised.

In regard to the *symptomatology of perforation of duodenal ulcer*, little need be added to what has already been said (at p. 85) in connection with gastric perforations. The great frequency with which neglected duodenal perforation simulates appendicitis should be borne in mind. Moynihan in 1901 collected 49 operations for perforation of the duodenum, in 18 of which the diagnosis had been appendicitis. If at an operation for appendicitis, especially in a male adult, no lesion of the appendix be found sufficient to account for the state of the peritoneal cavity, the surgeon will do well immediately to examine the region of the pylorus. In very many cases his search for a perforation will be rewarded.

Differential Diagnosis.—There are three affections which are frequently confused with chronic duodenal ulcer: gastric ulcer, chronic appendicitis, and gall-bladder disease. From *gastric ulcer* it usually may be distinguished by observing the long interval after food before the occurrence of pain in cases of duodenal ulcer; and by the occurrence of exacerbations in cold or wet weather, which are unusual if not entirely absent in patients with ulcer of the stomach. Meunier (1912) says that in gastric ulcer *immediate* relief of pain is secured by the ingestion of milk, while in duodenal ulcer this relief is delayed for 5, 10, or 15 minutes, and then occurs suddenly, and with belching of gas. Bolton (1913) points out that in duodenal ulcer, a heavy meal, especially of meat, staves off pain a long time; and when pain begins it lasts until the patient eats again, and eating at once relieves it. Gastric symptoms, he thinks (such as fulness, flatulence, eructations, etc.) favor the existence of gastric rather than of duodenal ulcer, as also does the only partial or occasional relief from pain by eating. Moynihan, on the other hand, as noted below, thinks flatulent dyspepsia is characteristic not of gastric ulcer but of gall-stone disease. From *gall-bladder disease* duodenal ulcer is to be distinguished: (1) by the regularity of the symptoms, occurring always at a specified time after food, and recurring as regularly, and being as regularly relieved by the ingestion of food; (2) by the complete absence of symptoms during

the intervals between exacerbations; and (3) by the fact that pain in duodenal ulcer is very seldom unendurable, while in attacks of biliary colic it frequently requires morphin for its relief. An acid dyspepsia, says Moynihan, indicates duodenal ulcer; while flatulent dyspepsia implies the presence of gall-stones. The differentiation from *chronic appendicitis* is very difficult; it seems at present that the mistake is more often made of thinking the patient has a duodenal ulcer, when the real trouble is chronic appendicitis, than *vice versa*. The symptoms of chronic appendicitis, however, do not bear such an invariable relation

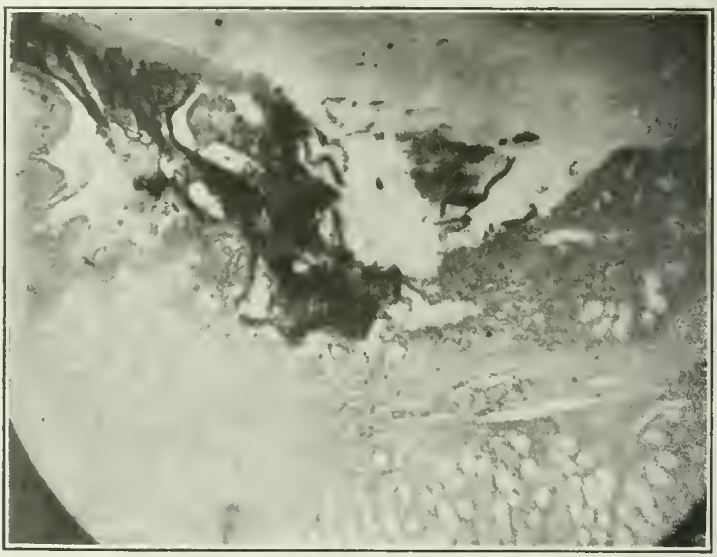

FIG. 54.—Microphotograph from Specimen of Duodenal Ulcer Excised from Case Represented in Fig. 55. This Section Shows the Ulcer still Ulcerating, though its Gross Appearance Indicated it was Healed. (*Dr. Reiman.*)

in time of appearance to the intake of food as do those due to ulcer of the duodenum, though they usually are brought on by indiscretions of diet; but they may develop without any apparent indiscretions in diet, especially following unusual exercise. Moreover, an exacerbation of appendicitis almost invariably keeps the patient to his bed for a few days, while in the exacerbations of duodenal ulcer this is unusual. Ingestion of food usually eases the pain of duodenal ulcer, while it will have no effect on that due to chronic appendicitis, or will make it worse. Moreover in chronic appendicitis there are no free intervals when dyspepsia is entirely absent for months at a time.

Prognosis.—Duodenal ulcer is a less serious disease than gastric ulcer only because of the great infrequency with which malignant changes occur in duodenal ulcer. Unless some treatment is applied

which will not only relieve the symptoms but entirely cure the disease, the patient is in constant danger of the acute complications (hemorrhage and perforation) which together occur in from 15 to 20 per cent. of cases. It is possible that modern medical treatment may do something more than secure latency of symptoms, but we know of no facts which render this certain. Should, however, a duodenal ulcer really heal under medical treatment, it is not probable that further trouble will be caused by obstruction from the cicatrix. Yet an ulcer which appears entirely healed on gross inspection may be shown by the microscope still to be unhealed (Figs. 54 and 55); while in rare instances hour-glass duodenum has developed (p. 203); and in most patients more or less crippling periduodenal adhesions will remain, interfering with evacuation of the stomach, distorting the gall-bladder, and rendering the patient's life miserable. On the other hand, surgical treatment offers for duodenal ulcer no less sure a cure than for ulcer of the stomach, and the operative mortality is as low. It has been possible to trace 60 patients with duodenal ulcer, operated on by the senior author during the years 1909 to 1914 inclusive. Of these 60 patients, 51 or 85 per cent. were ascertained to be free from symptoms one year or more after operation. Seven of the remaining 9 patients complained of nothing more serious than "occasional epigastric distress;" one had "persistent nausea" and one was reported "unimproved." Later statistics of cases of duodenal ulcer are included with those of gastric ulcer tabulated at p. 105. They indicate that approximately 90 per cent. of the patients traced have been found free from symptoms.

The prognosis when perforation has actually occurred is much less favorable. The actual figures collected by Robson, comprising 155 operations from various sources, gave a total mortality of 66 per cent., as may be seen in the following table:

Operations for Duodenal Perforation (1907)

Time	Cases	Deaths	Mortality per cent.
Under 24 hours	61	23	37.70
Over 24 hours	63	52	82.5
Not stated	31	28	90.30
Total	155	103	66.66

Fortunately at the present time perforations of the stomach and duodenum are recognized much earlier than was the case even a few years ago, and this factor, as well as more rational post-operative

treatment, has resulted in a considerable reduction in the mortality. The statistics of operations for perforations of the stomach or duodenum are tabulated at p. 122.

Treatment.—When a diagnosis of duodenal ulcer has been definitely made we believe it is a pure waste of time to postpone operative treatment. Delay is justifiable only when there is reasonable doubt as to the diagnosis. And when such a doubt cannot be dismissed within reasonable time under medical treatment, we believe it is the part of wisdom and of a sound mind to subject the patient to an exploratory operation.

FIG. 55. Duodenal Ulcer Cicatrizing, see Fig. 54. From a Patient in the Lankenau Hospital.

The best treatment, whenever applicable, is *excision* of the ulcer. If the ulcer is easily accessible, as is almost always the case if situated on the anterior or upper wall of the bowel, its complete removal by excision presents no difficulty whatever. When it is not easily accessible, its complete removal may be an operation of some difficulty, not to say danger, to one not thoroughly accustomed to abdominal surgery. If the surgeon does not feel himself competent, he should content himself with the performance of *gastro-jejunostomy*. In all cases this latter procedure is to be regarded as *an integral part of the operation for the cure of duodenal ulcer*. Coffey, Sherren and others indeed still consider gastro-jejunostomy alone as sufficient. Thus, Sherren (1920), with an experience in 389 operations for chronic duodenal ulcer (with only 9 deaths), adduces the following facts in support of his contention that gastro-jejunostomy alone is sufficient: in 8 cases he examined the ulcer *post mortem* at periods varying from 6 days to 9 years after gastro-jejunostomy, and in all (except that with only 9 days interval) the ulcer was healed; in 21 cases he examined the ulcer at a later operation, at intervals varying from 2 months to 10 years after the gastro-jejunostomy, and in all cases the ulcer had healed. He traced 348 patients for more than two years after operation for duodenal ulcer, and found 318 or 91 per cent. were cured. So he concludes that, unless the ulcer is adherent to another viscus, gastro-jejunostomy alone will cure it. But we believe that in the case of duodenal ulcers as in those affecting the stomach, the surgeon's first effort should be toward removal of the diseased area; and that gastro-jejunostomy,

(a) Duodenal Ulcer—Specimen Secured by Excision. Note Deep, Punched-out Crater, Without Evidences of Repair in Surrounding Duodenal Wall. Male, 25 years. Symptoms were those of Chronic Appendicitis, but Appendix when Exposed Found not Diseased Enough to Account for the Symptoms. Stomach then Exposed and this Juxta-pyloric Ulcer Found in Duodenum. Path. No. 9630. *Lankenau Hospital.*

(b) Duodenal Ulcer on Posterior Surface of First Part of Duodenum—Specimen Secured by Excision. Note Typical Punched-out Crater, with Threatening Perforation near its Centre. Male, 37 Years. Free from Symptoms for 1 Year after Operation, then Developed Symptoms of Chronic Cholecystitis, for which Cholecystectomy was Done $2\frac{1}{4}$ Years after Operation on Pylorus. Path. No. 9562. *Lankenau Hospital.*

Face p. 198

through indispensable as a supplemental operation, though effectual in preventing a recurrence, cannot always be relied upon to cure an already fully developed ulcer. The technique of excision of the duodenum is discussed at p. 367. Excision of isolated ulcers requires no special description.

Not only is it necessary to treat the duodenal ulcer at the time of operation, but it is necessary also to try to discover the focus of intra-abdominal infection which is to be regarded as the *fons et origo mali*. To this end the surgeon should also inspect the biliary tract, and if necessary drain the gall-bladder. The frequency of an accompanying pancreatitis should be recollected. An even more constant source of infection than the gall-bladder is the vermiform appendix, and unless there is some distinct contra-indication this obnoxious structure should be removed. This usually may be done through the same incision as that employed for exposure of the upper abdominal organs; but if it proves difficult to deliver the appendix through the upper abdominal incision it is better, we believe, to make a separate incision over the appendix rather than to lengthen unduly the upper incision.

When *perforation* has occurred, operation at the earliest possible moment is demanded. The sooner the surgeon opens the peritoneum, allows the extravasated matters to escape, and closes the perforation, the better it will be for his patient. If the duodenum is bound down by adhesions and the site of perforation is not easily accessible, a large sand pillow under the lower dorsal spine, as in operations on the biliary tract, will prove of considerable assistance in bringing the duodenum nearer the abdominal incision. The perforation should be closed with catgut or linen sutures. Usually a purse-string suture is sufficient for small perforations; while larger openings require a running suture placed either in the long axis of the bowel, or transversely, whichever proves easiest of application. If the sutures do not hold well, a tag of omentum should be stitched over the sutured area. Unless the peritonitis is extensive and the patient in a precarious condition, we advocate also in cases of perforation that the operation be completed by the performance of gastro-jejunostomy. This has been our practice for a number of years, and we believe our results justify our contention that gastro-jejunostomy promotes recovery (p. 127). In a few cases in which the duodenum was extremely friable and the perforation could be closed only imperfectly, resulting in temporary leakage of duodenal secretions and bile, we feel confident that the recovery of the patients was largely attributable to the existence of the gastro-jejunal anastomosis. This is

especially true in cases of subacute perforations of the stomach and duodenum unexpectedly discovered at operations undertaken for supposedly simple (uncomplicated) lesions. In such cases it may be impossible to make sutures hold in the region of the perforation, repeated attempts to do so only succeeding in enlarging the opening in the bowel; while resection is entirely out of the question. Here the patient's salvation depends on the performance of gastro-jejunostomy.

Retroperitoneal perforations of the duodenum usually make their presence known by the development of suppuration in the loin or flank, or occasionally in the right iliac fossa. The abscess should be opened where most accessible, and unless the discharge of duodenal contents is very free, a reasonable expectation may be entertained that the fistula will close spontaneously as is the case with most fecal fistulæ following the drainage of appendicular abscesses. Should the duodenal fistula show no tendency to close, however, in the course of four or five days, the surgeon should not delay too long to open the abdomen, establish a gastro-jejunal anastomosis and occlude the pylorus by plication of its anterior wall, or if necessary by section and closure of both ends. It is much better not to open the retroperitoneal tissues nor to make any attempt to close the perforation by suture.

Miscellaneous Affections of the Duodenum

Congenital Imperforation of the Duodenum, examples of which rare condition have been recorded by Cleemann (1874), Trump (1896), Stewart (1898), Weber (1910) and others, could be differentiated during life from imperforation of the pylorus only if the occlusion were below the papilla of Vater, thus allowing bile to be regurgitated into the stomach. Should a diagnosis be made, gastro-jejunostomy should be performed; but as malformations of the bile-tracts and liver sometimes co-exist, the prognosis is exceptionally gloomy. In the cases just referred to, the occlusion was below the papilla of Vater; in cases reported by Collum (1895), Emerson (1890), Hobson (1893), and others, it was situated above the entrance of the bile-ducts. A specimen of complete congenital occlusion of the duodenum, described by Keith (1910), is said to have come from the body of a child who lived until the age of 9 months.

A case of *congenital stricture of the duodenum* in a girl aged 13 days has been recorded by Shaw and Baldauf (1907); the lumen was found at autopsy to be permeable only to fluids under pressure. They quote

Kuliga as having collected 185 cases of congenital occlusion of the intestines: of these, 46 (25 per cent.) were of the duodenum, 94 of the jejunum, and 45 of the colon and rectum.

According to Terry and Kilgore (1916), there are on record about 70 cases of *congenital stenosis of the duodenum*. They report a case of duodenal stenosis in a man aged 24 years, which they regarded as congenital. The patient died from peritonitis after gastro-jejunostomy.

Acquired Stenosis of the Duodenum, of which Anders (1912) has collected 262 instances, is the result, in more than half the cases, of ulcer of the duodenum. Anders gives the following tabulation of causes:

Cause	Cases	Percentage
Ulcer of duodenum	140	53.44
Compression by root of mesentery	29	11.07
Carcinoma of duodenum	23	8.78
Carcinoma of pancreas	16	6.11
Sphincteric action of muscular layer of duodenum	15	5.73
Adhesions	12	4.58
Gallstones	9	3.44
Growths	4	1.53
Kinking	3	1.14
Cysts of pancreas	2	0.76
Miscellaneous	9	3.43
Total	262	100.00

Acute occlusions of the duodenum, apparently due to compression by the root of the mesentery, cannot well be separated from cases of acute dilatation of the stomach (p. 145).

The operation which has been adopted in cases of duodenal stricture by Bazy (1905), Mackenzie (1906), and others, is named *duodenoplasty*, and consists in longitudinal incision and transverse suture of the constricted portion of the bowel, in a manner similar to the pyloroplasty of Heineke-Mikulicz. If the adhesions are few or easily separated, and the duodenal wall not too thick or friable, this is a better procedure than gastro-jejunostomy in cases of stenosis due to changes in the intestinal wall. Of course if the stenosis is due to pressure from outside the lumen of the duodenum and if the pressure can be removed (gall-stones, pancreatic cyst), nothing further may be required. If the site of obstruction is inaccessible, as in the transverse duodenum, a duodeno-jejunostomy, as employed successfully by Frank (1913) in a child aged 11 months, is a more rational procedure than a gastro-jejunostomy, as in the latter case the bile and pancreatic secretions will have to traverse the stomach (retrograde through the pylorus)

before reaching the jejunum. Melchior (1914) adopted gastro-jejunostomy successfully in the case of a young man aged 19 years, who presented symptoms of duodenal obstruction below the bile papilla, found at operation to be due to a congenital abnormality; and he justifies the adoption of gastro-jejunostomy instead of duodeno-jejunostomy not only on account of the greater simplicity of the operation, but also because experience has shown that the duodenal secretions readily find their way through the anastomosis by way of the pylorus and are well tolerated by the stomach.

Chronic Dilatation of the Duodenum is occasionally seen. It is usually due to an obstruction at the duodeno-jejunal flexure or to

FIG. 56.—Hour-glass Duodenum from Cicatrizing Ulcer, Complicated by an Ulcer on the Lesser Curvature of the Stomach. (*From a Patient in the Lankenau Hospital.*)

pressure from the root of the mesentery. Bloodgood's observations (1912) tend to show that duodenal dilatation often is an accompaniment of redundant cecum and enteroptosis, the resulting tension on the mesentery occluding the duodenum where the latter is crossed by the superior mesenteric artery. He reports 4 out of 5 patients relieved of their symptoms by resection of the cecum and ascending colon. The symptoms of which these patients complained resembled those of pyloric stenosis, but there was bilious vomiting. Gregoire (1920) has also studied the subject, reporting 5 cases: he thinks recurrent attacks of bilious vomiting, with very marked exhaustion and sudden loss of weight, and without such pain as occurs in cholecystitis, is particularly characteristic. The diagnosis may often be made with reasonable

certainty if the fluoroscope is employed. He agrees with Bloodgood as to the etiological relation of congenital anomalies of the ascending colon but in some of his cases demonstrated that it was the taut middle colic artery and not the superior mesenteric which was responsible for the duodenal obstruction. He employed colopexy in his cases, in addition to gastro-jejunostomy. The same objections exist to gastro-jejunostomy alone in these cases as in those of stricture of the duodenum mentioned above. Yet Benjamin (1914) employed gastro-jejunostomy in a man aged 22 years, and reports freedom from symptoms nearly a year after operation. Stavely (1908) successfully adopted duodeno-jejunostomy, as have Quain (1920), Crouse (1920), and Ashhurst (1921), while Leriche and Sigaud (1912) report restoration of gastric function, by a gastro-jejunostomy with ligation of the pylorus to exclude the duodenum.

FIG. 57. — Duodenal Ulcer Cicatrized and Adherent to the Pancreas, Causing Hour-glass Duodenum. From a Patient in the Lankenau Hospital.

Christian has recorded (1907) a case where chronic dilatation of the duodenum was mistaken for hour-glass stomach.

There can be little doubt that many patients with symptoms of pyloric obstruction who are found at operation to have a patent pylorus are suffering from dilatation of the duodenum; this anomaly will be recognized by the surgeon if the possibility of the condition is kept in mind. If the cecum and ascending colon are congenitally mobile, they should be reattached to the posterior parietal peritoneum, as advised by Waugh (p. 69), and if this seems insufficient to relieve the condition gastro-jejunostomy or duodeno-jejunostomy should be done, together with suspension of the transverse colon to the anterior abdominal wall if this portion of the large bowel also is abnormally ptosed. Only in cases where the walls of the movable colon have become grossly thickened by long continued stasis do we believe colectomy will be indicated.

Hour-glass duodenum usually is a late result of stricture, following ulceration. The stricture usually is above the bile papilla, frequently quite close to the pylorus (Fig. 56). Mackenzie (1906) published details of several cases of this malformation, and suggests a number of operations for its relief. He has himself employed gastro-jejunostomy in two patients: cure resulted in the first case, which appears to have been a stricture rather than an example of true hour-glass deformity;

but the second patient, presenting a well marked deformity, though much improved by the operation, still had occasional gastric discomfort. Preferable operations are duodenoplasty (Ladevèze, Bazy, Mackenzie); duodeno-duodenostomy (Mayo); or gastro-duodenostomy to the distal pouch of the duodenum. All these procedures are much more easily executed after mobilization of the duodenum.

Eustermann (1914) reports 8 cases of hour-glass duodenum from the Mayo Clinic (1907–1913): posterior gastro-jejunostomy was done in 7 cases, and excision with plastic enlargement in the eighth patient. In one patient, under the care of the senior author at the Lankenau Hospital (March 27, 1913) (Fig. 57) posterior gastro-jejunostomy was adopted; unfortunately it has not been possible to trace this patient subsequent to his discharge.

Diverticula of the duodenum were described by Chomel (1710) and by Morgagni. Letulle (1899) noted in two cases little pouches in the neighborhood of the bile papilla, and thought they were to be explained as a congenital anomaly of development due to the budding out from the duodenum of processes, as in the formation of the liver and pancreas. This probably is the true explanation of most cases which are not clearly due to traction by adhesions. The subject was reviewed at length by Buschi in 1911, and by E. C. Moore in 1920.

Perry and Shaw (1893) classified diverticula of the duodenum as distension and traction pouches. Usually found close to the papilla, as in Letulle's patients, they are next most frequent at the pylorus. Usually they do not produce symptoms, and are found at autopsies in the aged. The development of pressure pouches is aided by the presence of the pylorus above and Ochsner's sphincter or compression by the root of the mesentery below. Traction diverticula are less usual, and generally are due to perigastric adhesions the result of ulcer or cholecystitis; they occur mostly in the first part of the duodenum. Perry and Shaw mention 14 cases of pressure diverticula, and found at Guy's Hospital records of three patients with traction diverticula. The *diagnosis* often may be made by the Roentgen rays (Cole and Roberts, 1920). Excision is the best *treatment*. Moore's patient was in perfect health $3\frac{1}{2}$ years after excision of the diverticulum.

CHAPTER IX

BENIGN TUMORS OF THE STOMACH AND DUODENUM

Benign tumors of the stomach and duodenum are rare. As has been seen in the preceding pages, a palpable mass, when not malignant, is almost invariably hyperplastic or inflammatory in origin. The non-malignant neoplasms most frequently encountered in the stomach are myoma, adenoma (including polyp), lipoma, and cysts.

Before proceeding to a detailed account of each variety, it will be well to state in a few words the general characteristics which most of these growths possess in common. Although found most frequently at autopsy, in patients who as a rule are not known to have suffered from gastric symptoms during life, it is evident that with the increasing number of operations on the upper abdomen, more of these tumors are now found at operation than formerly, even if they may not have been correctly diagnosticated before the abdomen was opened.

The patient is usually an adult, probably more often female than male, who has suffered from gastric indigestion for a number of years. Pain of a dragging and tearing character is sometimes a prominent symptom. Vomiting, if it occurs at all, usually arises a half hour or an hour after eating, but is present with no regularity as in gastric ulcer. The vomiting may occur only once or twice during the whole course of the illness, or it may, especially in the case of polypoid growths, recur whenever gastric peristalsis is excited. In very pronounced polypoid conditions of the mucosa, the nausea may be constant. Hematemesis is rare, being seen most often in adenomatous tumors, or in myomatous tumors which have penetrated the cavity of the stomach and have become ulcerated. The blood is then usually clotted before being vomited; the vomiting of bright red blood is quite unusual. The tumors in the course of time are prone to excite perigastsitis, and the adhesions both interfere with gastric motility, and cause increased pain. Frequently the tumor, though of fair size, is not palpable because held by adhesions beneath the costal margin, or because perigastritis renders the overlying muscles so rigid that satisfactory palpation is impossible. Large subserous tumors of the stomach with a long pedicle may, on the contrary, be very movable, and occasionally are found even in the

hypogastric region. The physical signs of hour-glass stomach may be simulated by submucous growths in the median portion of the stomach.

The long duration of a palpable mass is the chief means by which malignant disease may be excluded. A diagnosis of benign tumor is sometimes made in cases of inflammatory hyperplasia; and Kolaczek, as already mentioned (p. 188), diagnosed leiomyoma in a patient with a gastric diverticulum adherent to the anterior abdominal wall.

Myoma and Fibromyoma.—Myoma of the stomach was first observed by Morgagni. Steiner in 1898 collected 58 cases of myoma of the gastro-intestinal canal, 21 of which were situated in the stomach, and only 3 in the duodenum. Hake collected 59 additional cases in 1912 (this series included 3 cases of myoma of the appendix, and 4

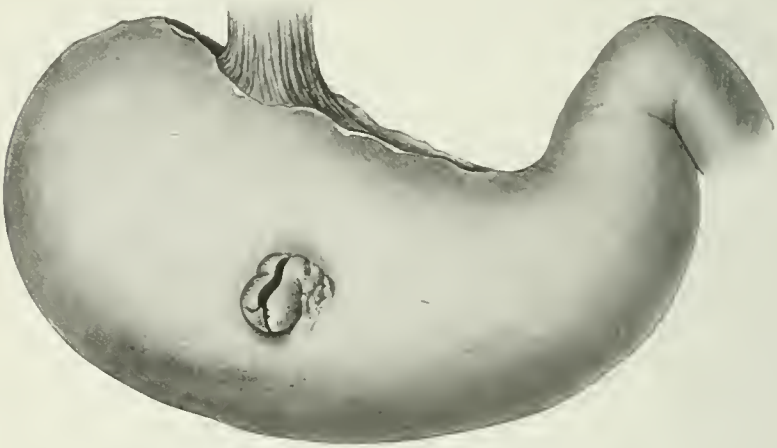

FIG. 58.—Fibroma of Posterior Wall of Stomach. One-half Natural Size. From a Specimen in the Museum of the Lankenau Hospital.

cases of myoma of the rectum). We have been able to find 52 further cases, recorded since the publication of Hake's paper. This makes a total of 125 cases of myoma or fibro-myoma of the stomach so that it can no longer be regarded as an excessively rare condition. It is probable, moreover, that very small myomata are sometimes overlooked at autopsy. Among 3500 autopsies at Genf, Tilger (1893) found 6 nut-sized myomata and fibromyomata of the stomach. Bircher (1908) recorded a case of pedunculated *fibroma* of the stomach.

Generally situated along one or the other curvature, they are less unusual close to the pylorus than at the cardia. Arising in the muscular tunic of the stomach wall, they grow either inward or outward, projecting as a rounded more or less nodular mass, beneath either the mucous membrane or the peritoneal surface of the organ. Being at

first attached to the muscular coat by a pedicle, this may become extremely attenuated, as in Cernezzi's case (1902), and finally all connection between the tumor and the muscular tunic from which it sprang may be lost (Virchow), as in the case of the analogous tumors of the uterus. Gastric myomata are almost invariably single if we except certain forms of polypus which are really more adenomatous or fibromatous in character.

Internal myomata, as those are called which project into the cavity of the stomach, frequently become ulcerated, and give rise to hematemesis or melena. Fatal hemorrhage occurred in cases recorded by Kemke, Miodowski and Niemeyer. These tumors are seldom very large, and often cannot be detected through the abdominal wall. External myomata, on the other hand, sometimes grow to an immense size, the tumor in Erlach's patient weighing 5400 grammes, and reaching in the case of Perls and Neelsen (1886) deep into the pelvis, and weighing 6000 grams. The gastric origin of such large tumors frequently is not recognized, both on account of their position in the abdomen, and because of secondary attachments which are formed to other structures. Yet adhesions to the anterior abdominal wall are unusual and free mobility is a frequent characteristic; especially noteworthy is the fact that the uterus can almost always be excluded as the seat of the disease. In 23 of the reported cases the tumor is recorded as being internal, in 21 as being external, and in 1 the growth was still interstitial in character when excised (Poirier, 1902) on account of pylorospasm. Of 25 patients 19 were over 40 years of age, and 13 of these were more than 50 years old. Of 27 patients whose sex is stated, 11 were males and 16 females.

The great majority of gastric myomata are strictly benign, but occasionally they are malignant. There is great confusion among pathologists as to the classification of these malignant varieties. In a great many myomata there have been noted certain areas of edema, amounting in some instances to myxomatous degeneration. When this condition is pronounced, the term myxomyoma is applied (Kemke). Cysts may be formed thus, or as the result of hemorrhage into the tumor. Certain cases have been reported (Goullioud and Mollard, Goullioud, Brodowski, Hansemann, Cohn, Shuyveninoff) in which gastric myomata, with more or less myxomatous degeneration, have given rise to metastases in the liver, peritoneum, and other structures; and in these metastatic nodules the primary growth was in large part reproduced, the cells being clearly smooth muscle cells, with an admixture of myxomatous, or degenerated cells. In their interpretation of the "degenerated" cells, either in the primary tumor or in the metas-

tases, pathologists are not in accord. They were long regarded as sarcoma cells, and the primary tumor was said to have undergone sarcomatous degeneration. But some writers, following the teaching of Bard of Lyons, and maintaining the theory of the specificity of tumor cells, claim that tumors such as those just described are pure myomata, and that the so-called sarcoma cells are nothing more than immature smooth muscle cells. These authorities (Parrot and Bérard; Devic and Gallavardin; Giuliani) name such a tumor Leiomyoma malignum. Steiner, Cernezzi, and others, distinguish between sarcomatous myomata, and myosarcomata; the latter being a malignant tumor *ab initio*, whereas a sarcomatous myoma is one which, though at first benign, finally undergoes malignant degeneration. Of course there is theoretically no reason why the connective tissue cells, present in a fully developed myoma, should not, as described by Steiner (1898), eventually become sarcomatous, just as they might do were the muscle cells themselves normal, thus forming a pure sarcoma. It is therefore theoretically possible for a myoma subsequently to become sarcomatous in this manner; but such a tumor, if it exist, would be more correctly named a sarcomatous myoma, or a myoma sarcomatodes, than a myosarcoma. And from our knowledge of pathological processes in general it appears hardly safe to conclude that muscle cells, even when already perverted into tumor formation, can subsequently, by metaplasia, become sarcoma cells. It seems to us more rational to look upon these myomata as being either sarcomatous tumors in muscular tissue (*myosarcoma ab origine*), or as being examples of leiomyoma malignum, as described by Devic and Gallavardin and by Giuliani. It may be mentioned in passing that similar tumors giving muscular metastases have been described in connection with the uterus.

Further confusion is added to the subject by the class of myomata in which angeiomatous changes exist. Some of these tumors are undoubtedly malignant, and are classed by some writers as angeiosarcomata, and by others as endotheliomata (von Bergmann; Nichols). Cyst formation is frequent in these angeio-myomata.

Magnus-Alsleben (1903) observed post-morten five patients with adenomyoma of the stomach, and he concludes that in these tumors the adenoma is the primary change, and that it is later crowded out, so to speak, by the myomatous overgrowth. Some authors have regarded certain instances of hyperemesis lactantium as due to a congenital myoma of the pylorus. (See p. 132).

In a case recorded by Monro and McLaren (1901), a pedunculated myoma near the pylorus was present in a stomach on whose lesser

curvature was a carcinoma. The two growths had no connection. Wade (1913) has recorded a case of intussusception of the stomach and duodenum into the jejunum, due to the presence of a pedunculated myoma near the pylorus. After reducing the intussusception the tumor was discovered and excised, the patient recovering.

Operative treatment should be undertaken as soon as a gastric myoma makes its appearance known. It is usually possible to remove the growth by resection of that part of the gastric wall from which it sprang; but occasionally formal excision (partial gastrectomy) is required.

In the first edition of this work we gave details of 14 operations for myoma or fibromyoma of the stomach, reported by various authors. Among these cases there were 8 recoveries and 5 deaths, the result in one case not being recorded. Since that date (1909) we have references to 31 additional operations of the same nature, 24 patients recovering, 1 dying and the result in 6 cases not being recorded. The whole series includes 38 terminated excisions or resections, with 6 deaths.

Adenoma and Papilloma.—Gastric adenomata are met with in two forms: (A) Sessile pedunculated growths, usually single, and practically indistinguishable from mucous papillomata; (B) Polyadenomata, or mucous polypi. Ebstein (1864) found 14 instances of gastric polyps among 600 necropsies.

(A) The former variety, which is usually understood when the term **adenoma** is employed, projects into the cavity of the stomach, usually in the pyloric region, in the form of a rounded, smooth or slightly lobulated tumor, evidently composed almost solely of hypertrophied mucous membrane. When solitary, such tumors have been known to grow to the size of an apple, or even to that of a fetal head at term (Chaput, 1895). Sklifossowsky (1898) recorded under the name of papilloma of the stomach two cases presenting much the same macroscopic and microscopic appearances as adenomata. Mauler collected all cases of adeno-papillomata recorded up to 1898. Hayem (1895) called attention to two cases of adenoma the structure of which resembled Brunner's glands, and which appeared to originate in the mucosa; the same condition has been since observed by other pathologists. These tumors usually rapidly penetrate the muscularis mucosæ, and proliferate in the submucous tissues. The more usual form of adenoma is strictly a mucous growth. It proliferates above the muscularis mucosæ, projecting into the cavity of the stomach; it very rarely becomes ulcerated, unless malignant, or unless it proliferates around the border of a gastric ulcer. It is usually single, but several

may be present in different parts of the stomach. In many patients the entire intestinal tract is involved.

When pedunculated, and with enough fibrous tissue in its pedicle to warrant the name of fibro-adenoma, this tumor forms one of the commonest varieties of gastric polypus. An intragastric polyp may occlude the pylorus, thus simulating pyloric stenosis from other and more frequent causes; and when easily displaced may produce intermittent dilatation of the stomach, as in a remarkable case reported in 1900 by Bennett (Figs. 59, 60). Gibson (1907) recorded a similar case. In one case quoted by Fenwick (1903) fatal *intussusception of*

Fig. 59.—Polypus of Stomach near Pylorus. (*Bennett.*)

Fig. 60.—Gastric Polypus near Pylorus, Acting as Ball-valve. (*Bennett.*)

the duodenum was produced by a polyp just below the pylorus. Wade's case of pedunculated myoma of the stomach, causing intussusception, has already been mentioned (p. 209).

In general it may be said that a fully developed gastric polyp is more apt to produce symptoms than is any other form of benign gastric tumor. Multiple polypi were found by Stevens (1896) at autopsy on a patient who had been subject to constant epileptic fits, with an aura arising in the stomach. Such cases are of interest in connection with the subject of gastric tetany. McCosh recorded (1900) a case of gangrenous gastritis from strangulation of a polyp in the stomach.

It is difficult to draw a boundary line between adenomatous changes frequently encountered in the stomach, in association with unhealed ulcers, and certain histological appearances which by some pathologists are considered pre-cancerous in nature. Ledderhose (1913) says Doening found gastric polyps associated with carcinoma in 24 out of 50 cases, and Versĕ in 22 out of 57 cases; he quotes Thorbeck as stating

that 50 per cent. show a tendency to carcinomatous change, and Wechselmann as claiming malignancy in from 50 to 60 per cent. of cases. For a further discussion of pre-cancerous changes the reader is referred to the chapter on Gastric Carcinoma (p. 248). The relation of myoma to adenoma, studied by Magnus-Alsleben, has already been referred to at p. 208.

(B) **Polyadenomata,** or **mucous polypi** of the stomach, are usually regarded as in some way the result of chronic gastritis. They are as a rule widely distributed over the interior of the stomach, transforming its surface into a pulpy mass, from which mucus can readily be squeezed. Each polyp is more or less distinct from the others, and none are larger than peas or small cherries. The centre part, or stalk, of each individual tumor is composed of fibrous tissue, containing blood vessels and lymphatics, and representing the normal submucosa. Over this core a thickened hypertrophied layer of smooth muscular tissue is found, corresponding to the muscularis mucosæ; while the free surface of the tumor, frequently cystic, is composed of hypertrophied and tortuous mucous glands. These dilated glands are said to resemble closely the uriniferous tubules in the cortex of the kidney. According to Ménétrier (1899), the cysts result from involvement of the ducts of the glands; whereas when the fundi of the glands are chiefly affected, the cysts are few in number and of small size. These mucous polypi are freely movable over the subjacent tissues, and the neighboring lymphatic nodes are never affected. Yet Norman recorded (1893) a case of polyadenomata, apparently belonging to this class, in which the change was not considered malignant although the glandular tissue had broken through the muscularis mucosæ and proliferated in the submucous tissue. Bier (1908) did gastro-jejunostomy for a similar affection (diffuse polyposis), the patient being reported as improved 18 months later, although microscopical examination of a portion of the gastric mucosa had shown "early malignant changes."

Ménétrier also describes a form of mucous polypus which he calls "*polyadenome en nappe*," in which condition the adenomatous formation is not confined to any circumscribed area or areas, with the formation of distinct polypi; but the hypertrophy and hyperplasia affect simultaneously all the glands over a fairly large area, or even through the whole stomach.

All these adenomatous tumors (the adenoma proper and the mucous polypi) have these distinguishing histological features: they are separated on the one hand from simple inflammatory or hypertrophic changes by the fact that although there is hyperplasia of the glandular

structures, yet the pepsin or oxyntic cells present in normal glands fail to be reproduced in the adenomatous neoplasms; and, on the other hand, they are distinguishable from adeno-carcinoma by the fact that nowhere may the epithelial cells of the adenoma be found to have penetrated the muscularis mucosæ or to be deprived of their normal basement membrane.

Symptoms.—Unless pedunculated, adenoma of the stomach is characterized by no very well defined symptoms. In cases recorded by Gourrand (1790), and by Quain and Beardsley (1856) a gastric polypus was vomited. In the case of mucous polypi the patient may complain of constant gastric discomfort, and nausea may be a prominent feature of the case. This condition has been suspected during life, and the suspicion confirmed at autopsy, in the case of patients who have suffered from a sensation of worms crawling around the stomach.

A correct diagnosis can rarely be made, even with the aid of skiagraphy; it is sometimes possible, however, to determine the presence of a benign tumor. In other case an exploratory operation is undertaken for the symptoms of pyloric stenosis, or even of prolonged gastric indigestion. Usually it has been possible to excise the tumor with or without partial resection of the gastric wall. Many more such operations are recorded for adenomata which showed malignant changes than for those which were undoubtedly benign.

Operations for the removal of adenomata or papillomata have been recorded by:

1. Lange (1892). Recovery.
2. Chaput (1895). Recovery.
3. Lyman (1896). Death in one month.
4. Bennett (1900). Recovery.
5. Robson and Moynihen (1904). Recovery.
6. Hinds (ibid). Recovery.
7. Wegele (1909). Recovery.
8. Wynhausen (1909). Recovery.
9. Sherren (1911). Recovery.
10. Khosroyeff (1912). Recovery.
11. Ponomarew (1912). Death.
12. Bruchi (1913). Recovery.
13. Myer (1913). Death.
14. Ledderhose (1913). Recovery.
15. Heinz (1914). Recovery.

16. Stoner (1914). Recovery.
17. Campbell (1915). Recovery.
18. Basch (Berg.) (1916). Recovery.
19. Basch (Roth) (1916). Recovery.

Lipoma.—These tumors may arise either in the submucous or the subserous adipose tissue. Small lipomata, the size of peas or beans, are not unusual. In such cases the masses of fat are probably not heterologous, but merely a localized increase in the amount of fat normally present. Randisi (1912) recorded a successful pyloroplasty for the removal of a nut-sized lipoma which had given rise to symptoms of pyloric stenosis. It is to the larger tumors, which may more justly be recognized as neoplastic in character, that this paragraph has special reference. Such cases have been reported by Cruveilhier (1835–42) (submucous); Russdorf (1867) (12 cases of subserous lipoma); Virchow (1867) (submucous); Orth (1887) (subserous); Murray (1888) (subserous); Tilger (1893) (2 cases of submucous lipoma); Fenwick (1903) (submucous).

Fischer reported (1905) a case of fibro-lipoma of the stomach in a woman aged 37 years, which had caused pain in the left epigastric region for about a month. An epigastric hernia developed, but there was no vomiting, no hemorrhage and no indigestion. The left rectus was rigid, and a diagnosis was made of recent inflammatory processes in a tumor of long standing, though no tumor was palpable. Operation showed the hernia to be an epiplocele, and the tumor was found on the lesser curvature of the stomach, not involving the mucous membrane. It was successfully resected; and microscopical examination showed a fibro-lipoma, with acute inflammation and hemorrhage into the substance of the tumor. The convalescence was delayed by five attacks of tetany on the seventeenth day after operation.

In none of these patients, except Fischer's, was operation undertaken; but as all the tumors were easily enucleated at autopsy, it would be perfectly proper to attempt their removal by gastrotomy, should their presence be discovered during life.

Myxoma.—According to Basch (1916) only three cases of gastric myxomata are on record, including that reported by Hansemann in 1895. They are properly regarded as degenerated forms of fibroma, etc.

Cysts.—Cysts are found in the stomach either as retention cysts of the mucous glands, or as the result of traumatism or the degeneration of other forms of tumor. There are also on record one case of dermoid

cyst and a few cases of hydatid cysts. The dermoid cyst (Ruyschius, 1737) contained hair, teeth attached loosely to bone, and other matters almost too wonderful for belief. According to Fenwick (1903), three of the hydatid cysts were found at autopsy; two (Bochlendorf, Barton) involved the stomach in the course of their development, but did not originate in the gastric wall; while in the case recorded by Castellvi y Pallares the gastric wall seems to have been the primary seat of growth. A fourth case of hydatid cyst has been recorded by Hartmann (1908); at the operation, by Dujarier, the cyst was found to have developed between the mucous and muscular coats of the stomach; it was successfully excised. Tuffier (1908) has operated on a patient with hydatid cyst developing in the gastro-hepatic omentum, and thus simulating a tumor of the lesser curvature of the stomach.

Numerous small cysts of the mucosa are frequently seen in certain forms of gastritis. They are true retention cysts, the inflammation obliterating the glandular orifices—a pathological change seen to an even more marked degree in the development of adenomata. These small retention cysts have practically no surgical interest, unless in connection with polyps or adenomata.

Traumatic cysts of the stomach are very rare. Zeigler (1894) and Chutro (1905) have each successfully operated on such a case. As shown in Chutro's valuable contribution to the literature of gastric cysts, the history is that of severe traumatism to the epigastric and left hypochondriac regions. Shock, pain, and persistent vomiting, but without evidences of peritoneal infection, are the immediate symptoms. A little blood may be vomited or passed from the bowels. After a few days or weeks, the more acute symptoms subside, though vomiting may persist; and the physical signs are more accurately localized to the stomach. A semi-fluctuating tumor may form. It is difficult to distinguish this from an encysted peritonitis; but the absence of suppurative signs will be an important clue. No time should now be lost in evacuating the contents of the cyst by laparotomy; suture of the cyst to the parietal peritoneum and drainage effected a cure in both patients. The cyst forms as the result of hemorrhage into the subserous (Zeigler) or submucous tissues (Chutro), aided perhaps by the effusion of gastric juice from the deeper portions of the mucous membrane, which in Chutro's patient was intact. The cyst, at first hemorrhagic, soon becomes serous or sero-purulent in character. The dangers of delay in operating are: (1) Infection of the cyst from stomach contents; (2) intraperitoneal rupture of the cyst, causing peritonitis; (3) rupture of the cyst into the stomach, creating a septic perigastric abscess. Any

of these events may be responsible for death in patients with gastric cysts not of traumatic origin.

More frequent are cysts which occur as the result of **degeneration** of other forms of tumors. According to Virchow, cyst formation is a not unusual termination of fatty tumors; and it is certainly true that most of the gastric cysts reported have contained fluid resembling disintegrated blood clot and fat. The large cyst surrounding the anterior wall of the stomach, found post-mortem by Read (1882) appears to have originated as a submucous lipoma which afterward underwent malignant change; while the remarkable case recorded by Hutchinson and Sloane (1856) in which a walnut sized cyst was found at autopsy, being both subserous and submucous, and its two sacs communicating by a narrow orifice in the muscular tunic, was possibly also of the same derivation, the contents being pinkish, thick, opaque, and glittering with plates of cholesterine. Albers (1862) observed a cyst of the lesser curvature at autopsy on a child. Hebb (1897) and Finnel (1874) have each recorded a case of gastric cyst which Fenwick classes as serous. In Hebb's case the cyst was lined with epithelioid cells, and may therefore have been lymphangeiomatous in origin. Finnel's patient also had carcinoma of the pylorus. Cases in which a probable hemorrhagic origin can be traced have been reported by Rendu, by Gallois, Hontang and Leflaive and by Anderson. Some of these so called hemorrhagic cysts may have been due to forgotten injuries, but it is probable that most of them were degenerations of pre-existing tumors. In the case recorded by Fenwick (1903) this origin is more certain in view of the lymphangeiomatous structure which is mentioned. A true lymphangeiomatous cyst has been reported by Engel-Reimers (1879) in whose patient the presence of an intensely deforming scar near the lesser curvature made it seem likely that the subserous cyst of lymphangeiomatous structure was not a true neoplasm, but merely a retention cyst due to the obstruction to the lymph channels by the cicatrix already described.

There remains to be discussed the extremely rare affection **"Gaseous cysts."** This disease, well known to the veterinarian from its frequent occurrence in swine, has been studied in its human relation by Hahn (1899), by Holstein (1899), and lately by Turnure (1913). According to the former authors the disease was first noted at autopsy, in 1754, by Duvernoy. Cloquet in 1820 reported a case of gaseous cysts (submucous and subserous) of the stomach and intestines. He had also observed it in the hog. Mayer in 1825 determined that the gas in these cysts was composed of oxygen and nitrogen, 15.44 parts of the former to 84.56 parts of the latter. Gaseous cysts of the vagina

and of the bladder have also been noted. Dupraz in 1897 found that the gaseous cysts of a stomach studied by him were dilatations of the lymph spaces, and that they were accompanied by chronic lymphangeitis. Three theories have been advanced to explain the development of these cysts: the *mechanical*, which assumes a leakage of gas from the lumen of the gut through some unperceived channel (the cysts being analagous to subcutaneous emphysema); the *bacterial*, which has nothing to support it except that the cysts have not always proved to be sterile; and the *neoplastic*, according to which certain degenerating cells of a tumor secrete gas. Hahn, Jaboulay, Vallas and Wendel have operated on patients with gaseous gastric cysts. Finney in 1908 published a paper based on a study of 19 collected cases of gaseous cysts of the gastro-intestinal tract; and Sloan (1920) refers to more than 50 cases now on record. Karsner, in Sloan's paper, supports the theory of the mechanical origin of these cysts, from some minute communication with the lumen of the gastro-intestinal tract.

Operative treatment has been undertaken in the following cases of gastric cysts:

1. Rendu (1880). Hemorrhagic cyst aspirated three times, and finally drained by a large canula. Death from peritonitis.
2. Gallois, Hontang and Leflaive (1884). Hemorrhagic cyst punctured twice. Death after many months from intraperitoneal rupture of the cyst.
3. Zeigler (1894). Traumatic cyst opened and drained. Recovery.
4. Winands (1895). Intestine punctured for obstruction. True condition found five years later at autopsy.
5. Anderson (1898). Hemorrhagic cyst opened and drained. Death in 24 hours of exhaustion.
6. Hahn (1899). Multiple gaseous cysts. Excision of those that were pedunculated, and puncture of others. Recovery.
7. Jaboulay (1901) explored abdomen of patient with gaseous cysts, did pylorodiosis by Hahn's method, and, believing the cysts were due to some low grade inflammatory process analogous to tuberculosis, closed abdomen without drainage and without interfering with cysts. Recovery.
8. Vallas (1901). Exploratory operation for intestinal obstruction with peritonitis. No cause found. Abdomen drained. Death. At autopsy innumerable subserous and submucous gaseous cysts of stomach and intestines. The submucous cysts had in some places caused intestinal obstruction.

9. Chuto (1905). Traumatic cyst opened and drained. Recovery.
10. Wendel (1911) successfully enucleated a cyst without opening the lumen of the stomach; examination showed the large cyst to be an accessory stomach and accessory pancreas.

Osteoma.—That it is not impossible for a gastric tumor to undergo osteoid transformation is proved by the unique case reported by Webster (1827). He found at autopsy on a patient who had died with symptoms of intestinal obstruction, that the pylorus was plugged as with a cork by a cartilaginous tumor, with numerous spicules of bone, which was adherent to the gastric wall near the pyloric orifice.

Concretions have been found in the stomach occasionally. They are generally due to the long continued use of mineral substances (bismuth, etc.) as medicines. Fenwick (1902) refers to four instances in which such concretions were composed of shellac or varnish. Hallas (1914) describes two further cases of shellac concretions in the stomach, discovered at autopsy. Both patients were inebriates and were accustomed to drinking furniture polish for the sake of the alcohol it contained. In the museum of the Academy of Natural Sciences of Philadelphia there is a remarkable specimen of a large gastrolith from a horse, deposited by Dr. John Ashhurst, Jr.

Angeioma.—Lammers (1893) reported a case of simple angeioma of the stomach, found at autopsy; there had been no gastric symptoms during life. Stokis (1905) at the autopsy on a twelve day old infant, who died in convulsions after profuse hematemesis and melena, found a capillary angeioma, in the submucous and muscular tissue of the stomach near the cardia, which was proved to be the source of the hemorrhage. Guisez (1913) detected an angeioma of the cardia by means of the esophagoscope. Treatment by dilatation followed by radium resulted in a complete cure. In connection with sarcoma and myoma angeiomatous changes are not unusual.

Lymphadenoma.—Gilly collected in 1886 51 cases of gastrointestinal lymphadenoma, the stomach being involved in 14 instances. A few cases have been recorded since. In all known cases, lymphomatous growths have been observed in other parts of the body as well in the spleen, lymph nodes, bones, pharynx, or intestines. In all cases of gastric lymphadenoma, the intestines were involved. This affection arises either in the subserous or submucous lymphatic tissues of the stomach. In the submucous tissues it exists either as a localized or diffused form, usually manifesting itself on the surface of the stomach by a polypoid condition of the mucosa. Ulceration is more usual in

the circumscribed form. The tumors which arise in the submucous tissues rarely cause obstruction, but those commencing in the subserous tissue, and which are usually diffuse, frequently penetrate, paralyse, and ultimately destroy the muscular coat, producing dilatation of the stomach and consequent stagnation of food. In some cases it appears to have been demonstrated that the disease originated in the neighboring mesenteric lymph nodes, and subsequently involved the subserous lymphatic structures of the stomach.

Hodgkin's Disease, affecting the stomach, deserves passing mention. In a case recorded by Scott and Forman (1916), no other region of the body was affected. A section of the stomach was removed at operation for study, and found to be histologically typical. The patient, a man aged 53 years, died two days after the exploratory laparotomy, about one year after the first symptoms of gastric disease.

Plastic Linitis.—This term was used by Brinton (1859) to describe a disease which had been previously studied, but had not been named, by Andral. It is an affection characterized pathologically as a diffuse sclerosis of the stomach, involving especially the submucous tissues, and accompanied by marked thickening of the gastric walls, and by a diminution in the capacity of the stomach. The process usually commences in the pyloric region, and gradually spreads, without affecting the mucous membrane, until the entire wall of the stomach becomes thickened and rigid, and its lumen much diminished in size. There have been many other synonymous terms employed to describe the same condition. Among the best known are: Cirrhosis or Fibromatosis of the Stomach; Leather-bottle Stomach; Zuckergussmagen; Submucous Sclerosis; Endogastritis Obliterans; Magenschrumpfung; etc. The causes to which this pathological change have been attributed are many. Carcinoma and syphilis sometimes produce a profuse gastric infiltration which even microscopically is distinguishable from this affection only with the greatest difficulty. Some cases of hyperemesis lactantium seem to be caused by an identical submucous sclerosis. Plastic linitis is usually regarded as benign, and is considered by most of those who have given most attention to the subject a special disease entity. The best articles recently published are those of Jonnesco and Grossman (1908), Kurt von Sury (1907) and Thomson and Graham (1913). Kurt von Sury concluded that cirrhosis of the stomach is due to the same cause as polyserositis, namely chronic passive hyperemia from cardiac insufficiency. Jonnesco and Grossman believe that it is simply a chronic inflammatory change, and in no way neoplastic in character. Thomson and Graham, from their study

of 50 specimens, conclude that it is a disease *sui generis*, chronic inflammatory in nature, caused by ulceration or by ulcer, and frequently overgrown by carcinoma. Histologically it appears to be nearly related to the lymphadenomatous changes just described. Its connection with chronic obstruction of the efferent lymph vessels of the stomach does not appear to have received sufficient attention. Its relations with endothelioma and sarcoma are not clear.

By microscopical examination it is sometimes impossible to say whether the epithelioid cell nests which infiltrate the submucous and

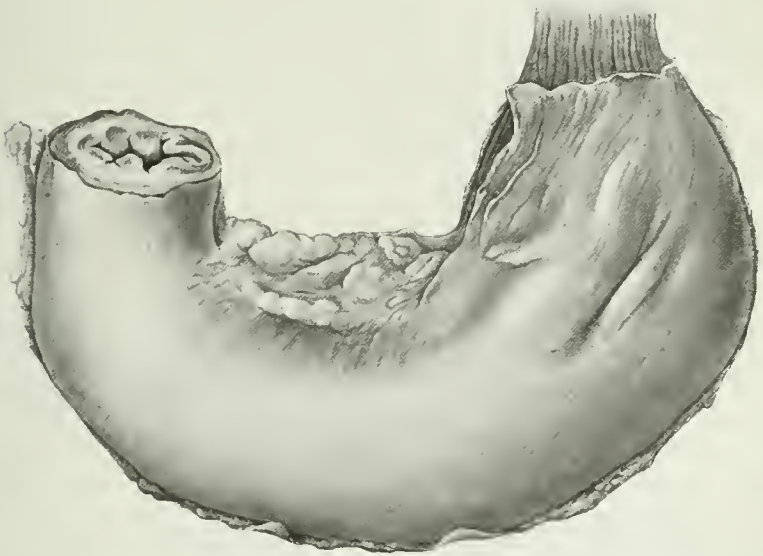

FIG. 61.—Total Contraction of the Stomach (Magenschrumpfung) from Plastic Linitis. Natural Size. From a Specimen in the Museum of the Lankenau Hospital.

muscular tissues are really epithelial in derivation (carcinoma), or due to proliferation of previously existing endothelial cells lining the lymph channels which normally exist in these situations (see Jaboulay's case (1905) of subtotal gastrectomy for an infiltrating growth thought by Gayet and Patel to be epitheliomatous). Porter (1915) quotes W. J. Mayo as stating that "they have had in their clinic perhaps 20 cases of so-called leather-bottle stomach, and all those they have been able to excise and make sections of proved to be malignant although sometimes many sections had to be cut before a malignant area was found."

As the question of the pathology of this affection is still *sub judice*, it is probably safer at present to regard it as a pathological change

which may arise in several different conditions—ulcer, carcinoma, syphilis, polyserositis, lymphatic obstruction, etc.

Treatment must usually be palliative. Should the change be recognized early enough, *partial* or *subtotal gastrectomy* should be attempted. In a somewhat similar case Sheldon (1906) did *gastro-jejunostomy*, and reported his patient in good health three years and a half later. If the disease has progressed so far as practically to obliterate the cavity of the entire stomach, *duodenostomy* should be done; or if the disease has invaded the duodenum, jejunostomy may be the last resort. This operation was adopted in one case of this disease by v. Eiselsberg (1908), with gratifying result, the patient still using the fistula with comfort five years after the operation. We have found it impossible to collect reliable stastistics as to operations for this condition, as many surgeons report as instances of plastic limits any cases of gastric carcinoma in which the stomach walls are much thickened.

The senior author has had under his care six patients with diffuse fibrosis of the stomach clinically resembling plastic linitis. The history of one patient is as follows:

Frank K. aged 46 years. Two uncles died of phthisis. When 22 years of age the patient was accidentally inoculated with syphilis, developing a chancre on the thumb. Received antisyphilitic treatment for 2 years. In 1903, three years before his admission to the German (now Lankenau) Hospital, he had an attack of pyelitis, after the passage of a urethral sound. Following this illness he suffered from acute gastritis, having hiccoughed for 11 days. For the last 2 years has suffered from pain and tenderness in the epigastrium. He vomited only when he forced himself, and then brought up food taken one or two days previously. No hematemesis or melena. For past 7 months no food but milk. Has lost 100 lbs. in weight. Says he cannot retain more than 6 oz. in his stomach at one time. Physical examination was negative except for tenderness and rigidity in the epigastric region. Examination of stomach contents showed: total acidity, 10; no free hydrochloric acid; no lactic acid; no Oppler-Boas bacilli. Bloodcount: R. B. C., 3,410,000; W. B. C., 8,000; Hb., 62 per cent.; color index 0.8.

Operation June 3, 1906. The stomach was found much contracted, its walls dense and fibrous, and resembling in appearance and shape the small bowel. Posterior gastro-jejunostomy was done, the gastric wall being an inch in thickness. Recovery was uneventful, and the patient remained in good health for 2½ years after the operation, but has since died, of unknown cause.

In five other cases the operation consisted merely in exploration, the stomach's condition not warranting any operative relief. One of these patients was later operated on in another city, a jejunal fistula being established; after death, which occurred a few weeks subsequently, the lesion was proved to be carcinomatous.

CHAPTER X

MISCELLANEOUS AFFECTIONS OF THE STOMACH AND DUODENUM

Tuberculosis of the Stomach is rare. Broders (1917), in his admirable review of the subject, reached the conclusion there were only 49 positive cases on record, 118 probable cases; while he classed 59 of the cases reported as such as "doubtful," and entirely rejected 80 of the reported cases. According to Curschmann (1904), in 900 autopsies on tuberculous subjects Durk found the stomach involved only in 4 cases; and in 2000 autopsies Simmonds found only 8 tuberculous ulcers of the stomach; while Glaubitt, according to Barchasch, noted 47 tuberculous ulcers in autopsies on 2237 tuberculous patients. Gossmann (1912) reports 18 cases of ulcerative gastric tuberculosis among 5900 autopsies, or 0.31 per cent. of the total autopsies, and 0.76 per cent. of those (2360) which showed tuberculosis in any situation. Adler (1907) found 11 cases of tuberculous gastric ulcer in 839 autopsies on tuberculous children; which makes it appear rather less unusual in children than in adults.

According to Ricard and Chevrier (1905), Louis, in 1825, first recognized tuberculosis of the stomach; but Hattute (1874) was the earliest to note pyloric stenosis from tuberculosis; and it was not until 1894 that the subject was brought prominently before the profession by Durante.

It is important, in the first place, to distinguish between gastric ulcers occurring in tuberculous patients, and those ulcers due to the local action of the tubercle bacillus. The former may be of the ordinary type ("round," "acute," "chronic," etc.) or in some instances may be due to the toxemia of tuberculosis localized in other parts, the ulcers thus resembling in origin those erosions on which Dieulafoy and more recently his pupil Gandy have laid such stress, as caused by the toxemia of various infectious diseases. If the toxemia of tuberculosis give rise to mucous erosions in the stomach, these erosions may later become infected with the tubercle bacillus, either ingested with food, or swallowed with the sputum. Or a true tuberculous ulcer may possibly arise *de novo*, without the previous existence of an erosion or an open ulcer. Secondary infection of an already existing gastric lesion is probably much the more frequent origin. The portal of

entry is usually through the gastric mucous membrane, from the cavity of the stomach; though infection by the blood and lymph streams is also recognized as possible. The rarity of the affection is no doubt due to the short time that the ingesta normally remain in the stomach, as well as to the antiseptic action of the gastric juice. Certainly intestinal tuberculosis is much more frequent than is gastric (Barchasch says the intestines are involved in from 47 to 63 per cent. of phthisical patients who come to autopsy); and gastric tuberculosis when it does occur is in the immense majority of cases secondary to some tuberculous lesion in other parts of the body, particularly the lungs or bronchial lymph nodes. According to Van Valzah and Nisbet, "sometimes the only detectable local lesion is tubercle of the choroid or a laryngeal or nasal ulcer." They also remind the reader that the tubercle bacillus may be found in the urine when it is absent from the sputum. Barchasch (1907) admitted six cases as examples of undoubte primary tuberculosis of the stomach but Broders (1917) claims no case can be recognized as primary in the stomach if there are tuberculous lesions in any other part of the body; and states that no case so far reported meets this requirement.

The tubercle bacillus may pass through the gastric mucosa, leaving it intact, and lodge and proliferate in the neighboring lymph nodes. This is very unusual. When the lymph nodes have been long involved, whether primarily or secondarily diseased, they become caseous and sometimes calcareous. Those along the lesser curvature may soften and rupture into the cavity of the stomach; but at the pylorus the thickness of the wall is so great as to prevent this termination. Peripyloric tuberculous lymphatic involvement is sometimes a cause of pyloric stenosis without lesions of the gastric mucous membrane.

Poncet and Leriche (1908) distinguish three main forms of surgical tuberculosis of the stomach: an ulcerated form, which is rendered surgical only by its complications; an hypertrophic form, the gastric tuberculoma, simulating carcinoma; and finally an inflammatory form, which differs from other forms of gastritis only in its etiology.

Gastric tuberculosis is almost always ulcerated. The disease affects by preference the submucous and subserous tissues, the muscular tunic escaping as a rule. But in the pyloric region, where the disease usually assumes the hyperplastic form, the muscular coat is prone to invasion. In the body of the stomach tuberculosis is usually diffuse. The ulcers are ragged, undermined, leaving free overhanging edges of mucosa; and when of long duration are seated on characteristic raised and thickened bases, called by the French "*remparts*." The

ulcer often assumes the transverse character, parallel with the blood vessels, as are the similar ulcers of the intestine. Perigastritis usually occurs in time to protect against perforation into the abdominal cavity; and hemorrhage also is rare. In some cases the duodenum has been opened by ulceration, forming a gastro-duodenal fistula. This may temporarily relieve the symptoms of pyloric stenosis. The colon has also been penetrated by the ulceration of a tuberculous gastric ulcer. Perforation of the esophagus by a tuberculous ulcer was the cause of death in a patient of Giorgi, who was found at autopsy also to have had tuberculous ulcers of the stomach.

Among the 107 cases of gastric tuberculosis studied by Ricard and Chevrier there were only three in which no other portion of the digestive tract was involved in tuberculous disease. The small intestine, the mesenteric lymph-nodes, and the cecum are oftenest affected.

The **symptoms** are those of gastric ulcer. Perforation also is rare: Ricard and Chevrier (1905) report one case, as do Rènon and Verliac (1907); both patients died. Pyloric stenosis from tuberculous hyperplasia is one of the usual forms of the disease. When present, it is not difficult to detect the stenosis, by the usual symptoms and physical signs; but it is only by a searching examination for tuberculous lesions elsewhere in the body that the etiological diagnosis of the gastric lesion can be made. The tuberculin test may aid in determining the question. At operation it may be difficult to distinguish these cases from those of pyloric carcinoma. Tuberculosis is less unusual in those under 30 years of age. Inflammatory hyperplasia, plastic linitis, and even syphilis have to be considered.

The **prognosis,** so long as there is no pyloric stenosis, depends rather upon the other tuberculous lesions in the body than upon those in the stomach. If pyloric stenosis is present, the prognosis is absolutely bad, without operation.

The **operative treatment** of gastric tuberculosis has so far accomplished little beyond relieving the most distressing symptoms and moderately prolonging life. Zesas (1913) refers to 8 gastrectomies, with 4 deaths; and among 23 gastro-jejunostomies to which we have reference there were 4 more or less immediate deaths, 9 patients who recovered temporarily but died within a few months either from local or general tuberculosis, and only 10 who recovered and lived long enough to profit from the operation. One patient survived for three and one-half years after gastro-jejunostomy and then died from an abscess of the liver, the exact cause of which could not be determined at autopsy (Chevassu, cited by Ricard and Chevrier, 1905).

Operation is not advisable except to relieve pyloric obstruction. If it can be determined that other tuberculous lesions in the body are not such as to render the expectation of life unreasonably limited, it may be proper in selected cases to undertake exploratory laparotomy with a view to prolonging life by improving nutrition through the means of some palliative operation. Ricard and Chevrier severely condemn all attempts at excision as futile and extremely liable to disseminate the tuberculous process. If, however, the disease is limited to the pylorus, and especially if the tuberculosis appears primary in the stomach, partial gastrectomy should be preferred. In other cases the nature of the operation will depend upon the local conditions and upon the extent of constitutional involvement. Gastrolysis may be sufficient when the pyloric obstruction is caused by perigastric tuberculous peritonitis. In most cases gastro-jejunostomy is the operation of choice. In some patients pyloroplasty may give satisfactory results.

Syphilis of the Stomach.—It has been said that the stomach is affected in about one per cent. of syphilitic patients. Among 243 autopsies which showed unmistakable lesions of syphilis, Chiari found (1891) syphlitic lesions of the stomach in three cases. Morgan (1915) reports that by employing the Wassermann test in all patients, complaining of severe gastric symptoms he found only 1 per cent. infected with syphilis. In a series of 7545 gastric cases Smithies (1915) found 1.6 per cent. had syphilitic lesions of the stomach. In 1898 Flexner was able to collect only fifteen authentic instances of gastric syphilis, including one of his own. During the last two decades the attention of surgeons as well as physicians has been more particularly directed to the stomach, and numerous other observations have been published, there being now on record probably about one hundred cases of gastric syphilis.

As in the case of tuberculosis it is important to distinguish ordinary ulcers of the stomach occurring in syphilitic persons, from gastric lesions primarily due to the syphilitic virus. In determining the true nature of the lesion, the microscopical appearances are a surer test than is the result of specific treatment. There is very good reason to believe that anti-syphilitic treatment will favorably influence the course of non-specific gastric lesions in syphilitic subjects, by improving the general health; and therefore it should not be assumed that the gastric lesions are specific merely because a course of mercurials or iodides prescribed for a syphilitic patient is followed by subsidence of the gastric symptoms. Yet it must also be remembered that in the

interpretation of histological appearances even professed pathologists are not always in accord, and that this is particularly the case with syphilitic as well as with cancerous lesions.

Syphilis affects the stomach either as a diffuse *infiltration*, invading especially the submucous tissues, or as a distinct tumor, a *syphiloma* or *gumma*. In either case the lesions are prone to ulcerate, as a result of the endarteritis and consequent interference with the nutrition of the overlying mucosa. Yet hemorrhage, unless occult, is an infrequent sign. In Flexner's patient, whose stomach was the seat of an ulcerated submucous gummatous infiltration, death occurred from perforative peritonitis. In other cases, stricture may result. More common as a cause of obstruction is the localized syphiloma. Bird (1907) claimed to have observed 12 cases of gastric syphiloma, the pylorus being the the seat of the tumor in 11 of the patients. Downes and LeWald (1915) who studied 8 cases in which, in the course of two years, the diagnosis of syphilis of the stomach was made with a fair degree of certainty, lay stress on the constancy of the pain, which they describe as gnawing in character, and which is opposed to the periodicity of the painful attacks of simple gastric ulcer. They regard a rather dumb-bell shaped hour-glass stomach as revealed by the X-ray as typical of syphilitic disease.

Syphilitic gastritis, in which there may be no specific lesions of the stomach, is a fairly frequent accompaniment of the disease. It is often encountered in patients with hereditary syphilis. It is sometimes caused by anti-syphilitic treatment.

The **diagnosis** of gastric syphilis rests on three points: first, the history of syphilis in the individual patient or the presence of a positive Wassermann reaction; second, the resistance of the gastric symptoms to all ordinary remedies; and third, the rapid amelioration under specific treatment. But even though all three of these postulates be fulfilled, the gastric lesions may not be due to a local manifestation of the syphilitic virus; and even though one or more of these factors be wanting, the lesions of the stomach may yet be syphilitic.

Operative treatment is demanded only when one of the gastric orifices is obstructed by a syphiloma which is unaffected by specific treatment. Morgan (1906) reported the case of a patient who had suffered for four years from anorexia, flatulence, thirst, and dilated stomach with pyloric obstruction; these symptoms were attended by loss of weight, progressive emaciation, and finally the development of a palpable

mass in the pyloric region. As syphilis was denied, a diagnosis of carcinoma was made; but the patient finally acknowledged having having had a chancre, followed by secondary lesions, four years previously. Iodide of potash was administered, the symptoms were promptly relieved, and health was restored. Tuffier in 1899 did gastro-jejunostomy in a syphilitic negro, whose pylorus was obstructed by a large, firm, elastic tumor which disappeared four months after the operation. In the series of 8 cases reported by Downes and LeWald, gastro-jejunostomy was done in 5 because of obstruction. The 3 patients without obstruction were rapidly relieved by anti-syphilitic treatment. It is only rarely that excision is to be undertaken. If any operation is requisite to aid medicinal treatment, some palliative procedure is to be preferred: gastro-jejunostomy for pyloric stenosis and gastrostomy for obstruction of the cardiac orifice. Of course if the tumor is possibly, malignant, and is operable excision should be done. Bird and others lay much stress on the hepatic and peritoneal involvement as characteristic of syphilis; Bird considers of importance the presence of "bluish striæ following the course of the lymphatics, or of splotches of opaque bluish white on the serosa or starred cicatrices with strongly fibrous or even calcareous centers." Hausmann (1911) thinks fixed pyloric tumors that do not cause stenosis should make one suspect syphilis especially if the diseased area is dilated rather than contracted.

Unless there is very good reason to suspect a syphilitic origin for the gastric symptoms, the surgeon will best consult the interests of his patient by not delaying too long an operation which is clearly indicated, for the sake of trying the effect of anti-syphilitic remedies. He should bear in mind, moreover, that symptoms of gastric distress in a syphilitic patient are frequently caused by the ingestion of anti-syphilitic remedies; and should such a cause for the symptoms be probable, these remedies should be discontinued temporarily. On the other hand, should the syphilitic origin of the gastric lesions become manifest by operation or otherwise, no time should be lost in getting the patient under the influence of mercury and the iodides: such treatment will be an important adjuvant to any operation that shall have been performed. Permanent cure, however, as noted by Smithies (1915), can hardly be promised, as the gastric symptoms show a persistent tendency to return even after long periods of latency.

We have references to the following operations for syphilitic lesions of the stomach:

Operation	Cases	Deaths
Exploratory	5	0
Gastro-jejunostomy	11	3[1]
Partial gastrectomy	7	0

Phlegmonous Gastritis.—Phlegmonous gastritis is a rare form of inflammation of the stomach, which has been recognized, according to Schnarrwyler (1906), since 1656, when a case was first observed by P. Borel. Jensen (1911) collected 131 cases of the condition. It has been described under a multitude of names, which are given at length by Leith (1896), and of which the most frequently employed are "submucous gastritis" and "suppurative linitis."

The disease is defined by Schnarrwyler as a "diffuse purulent inflammation of the stomach, which has its chief seat in the submucosa, but which may later produce a lymphy and finally purulent infiltration of the intermuscular connective tissue, and thus eventually reach the serosa; while on the other hand the overlying mucosa becomes infiltrated with pus cells and swollen."

Robson and Moynihan, in their work on Diseases of the Stomach (1904), went into the pathology and symptoms in considerable detail, and little can be added to what they then wrote. According to the definition given above, only diffuse submucous inflammations should be included; but as there is no doubt that well localized phlegmons of the gastric wall are occasionally encountered, it seems scarcely worth while to make a separate classification for "phlegmon ventriculi," and we therefore agree with Robson and Moynihan, who describe phlegmonous gastritis as existing in two forms, the circumscribed and the diffuse.

Although there seems good reason to believe that the disease occasionally arises without any macroscopical lesion of the gastric mucosa, it is more frequently encountered as a complication of gastric ulcer, or a sequel to some operation on a stomach which is already the seat of catarrhal gastritis. The streptococci are the micro-organisms most often found; but staphylococci, colon bacilli, and even gas-bacilli, have been recovered from the stomach in some instances.

Operations have proved the exciting cause in cases recorded by Schnarrwyler, Eiselsberg, Page, and others. In Schnarrwyler's patient

[1] Two deaths occurred several weeks after operation.

an anterior gastro-jejunostomy had been done by Hildebrand for an inoperable mass obstructing the pylorus. Death followed in five days; and the autopsy showed that it was caused by a diffuse sero-purulent peritonitis arising in a purulent infiltration of the stomach walls, which had not been present at the time the operation was done. In v. Eiselsberg's patient death from phlegmonous gastritis followed six days after he did "gastro-enterostomia retrocolica anterior;" and in a patient operated on by Page, fatal phlegmonous gastritis followed the performance of gastrostomy for stricture of the esophagus.

The clinical picture presented by a patient with diffuse phlegmonous gastritis is thus graphically summarized by Robson and Moynihan: It is that "of a patient acutely ill from some febrile disease, with irregular elevations of temperature, very feeble and rapid pulse, vomiting, constant pain in the abdomen, referred generally to the epigastrium, and slight tenderness on deep palpation. It is therefore," they proceed, "not a matter of surprise to learn that a positive diagnosis of phlegmonous gastritis has never been attempted."

The purulent collections in the submucosa are solitary or numerous; the abscesses vary in size from that of a millet seed to that of a man's fist; they may perforate either into the stomach or the abdominal cavity; and in either case are almost surely followed by death. According to Novak (1919) less than 25 authentic cases of circumscribed phlegmonous gastritis (submucous abscess) are on record. Whether the disease be of the circumscribed or of the diffuse form, peritonitis without macroscopical perforation of the gastric wall will be the nearly inevitable result; and unless exploratory operation were to be undertaken on very indefinite symptoms, peritonitis will have developed before a diagnosis is made.

In the circumscribed form of the disease it is possible to evacuate the abscess (Bovee, 1907) by operation or even to excise the portion of the stomach affected (Kayser, 1911, Bircher, 1912, Novak, 1919); but little can be done for the diffuse phlegmonous inflammation. Possibly by isolating the stomach with sterile gauze and incising its walls down to the mucosa, or even by opening its cavity widely, a favorable issue might be anticipated, if the operation were done before general peritonitis supervened. Five patients are said to have recovered without operation, but in these the existence of phlegmonous gastritis was only inferred.

Operations on patients with phlegmonous gastritis have been performed by:

1. Leith (Edinburgh Hospital Reports, 1896, iv, 51). Patient presented symptoms of diffuse purulent peritonitis. A median hypogastric incision seemed to show that the inflammation was more acute in the right iliac fossa. A second incision was therefore made, and the appendix removed. It did not appear to be gravely diseased. The abdomen was irrigated, the wounds were closed, but the patient died in seven hours. Autopsy showed that the peritonitis arose from diffuse phlegmonous gastritis, and that the inflammation had probably spread from the stomach first to the right iliac fossa (as is frequently the case with patients with perforated duodenal ulcer), and had subsequently become generalized.
2. Lennander (Lengemann: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1902, ix, 762). Patient with diffuse epigastric peritonitis. The stomach seemed to be the original seat of the disease, and it was tamponaded. Death in 60 hours. Phlegmonous gastritis found at autopsy.
3. Mikulicz (Lengemann: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1902, ix, 762). Patient with symptoms of perforated gastric ulcer. Operation disclosed sero-purulent peritonitis around stomach, from a not well localized phlegmon of the gastric wall. No perforation found. Irrigation and drainage. Recovery.
4. Bovée (Trans. Southern Surg. and Gyn. Assoc., Dec., 1907, in Jour. Amer. Med. Assoc., 1908, i, 311). A case of circumscribed suppurative phlegmonous gastritis; recovery after gastrostomy.
5. Adams (Lancet, 1910, i, 292). A case of acute primary phlegmonous gastritis in a girl aged 13 years. Exploratory laparotomy. Death. Peritoneal fluid yielded a pure culture of the pneumococcus.
6. Clarke, Hertz, and Rowlands (Guy's Hospital Reports, 1910, lxiv, 295). Case due to swallowing hydrochloric acid. A mucous cast of the pyloric half of the stomach was vomited. Gastro-jejunostomy. Recovery.
7. Kayser (Deutch. med. Woch., 1911, xxxvii, 631). Partial gastrectomy. Recovery.
8. Bircher (Corr. Bl. f. Schw. Aerzte, 1912, xlii, 303). Partial gastrectomy and cholecystectomy (duodenum and gall-bladder involved as well as stomach). Recovery.

9. Novak (Jour. Am. Med. Assoc. 1919. lxxiii, 1038). Partial gastrectomy for pyloric tumor, arising from lesser curvature and bulging, from a sessile base 6 cm. long, about 3 cm. into the cavity of the stomach.

Operations for Phlegmonous Gastritis

Operation	Cases	Deaths
Exploratory	4	3
Gastrostomy	1	0
Gastro-jejunostomy	1	0
Partial gastrectomy	3	0
	9	3

Volvulus of the Stomach.—Volvulus of the stomach appears to have been observed at least in 35 cases. In ten cases (Bourcart, Hahn, Hedlund, Hermes, Kocher, Langerhans, Mazotti, Niosi, Saake, Schüler) hour-glass contraction of the stomach seems to have acted as a predisposing cause, though it is doubtful whether in Saake's patient a true volvulus existed. In other cases no cause was demonstrated, but in some there was noted a marked lengthening of the gastric ligaments, and in others (Berg, v. Haberer) a tumor near one of the orifices may have excited undue gastric peristalsis. The symptoms are usually pain in the epigastrium, but without fever or evidence of acute peritonitis. If the cardia be occluded by a twist, there will be no vomiting, and introduction of the stomach tube will be difficult or impossible; if it be not occluded, vomiting will be persistent; and the absence of bile from the vomitus may be an indication that the pylorus is occluded. The symptoms are those of intestinal obstruction and the physical signs closely resemble those of acute dilatation of the stomach; indeed as the usual effect of the volvulus is to occlude both orifices, dilatation of the stomach naturally follows. As already remarked, it is very difficult or impossible to introduce a stomach tube; and this fact alone shows that something more than mere gastric dilatation exists.

The rotation may take place in any direction, though there are three more or less typical directions in which it usually occurs. These are: (1) Around an antero-posterior axis, in which case the stomach rotates either "clockwise" or "contra-clockwise" as viewed from the front; (2) around a transverse axis in the frontal plane, in which case the stomach rotates as an advancing or as a retreating wheel, viewed from the front of the body; or (3) around a longitudinal axis

in the sagittal plane (an axis more or less at right angles to the greater curvature), when the stomach revolves either clockwise or contra-clockwise when viewed from the head of the patient. The most frequently encountered form is volvulus around a transverse axis in the direction of a retreating wheel (21 cases in all). In most instances the transverse colon followed the stomach upward and backward, being found between the stomach below and the liver and diaphragm above. In Dujon's patient the great omentum, except at

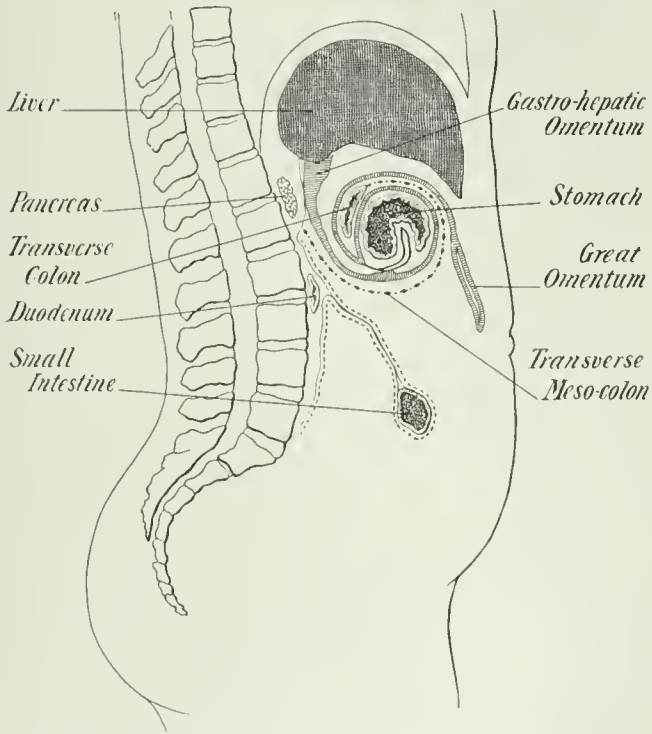

FIG. 62.—Wiesinger's Case of Volvulus of the Stomach.

the pylorus, and the gastro-splenic omentum were torn off from their gastric attachment by the volvulus. He found that he could not produce this form of volvulus in the normal cadaver, unless the gastro-splenic and great omenta were ruptured. In Borchardt's patient the transverse colon maintained its normal position, but the gastro-colic omentum, which was very broad, was stretched to its utmost. In most cases the spleen has been more or less displaced; it may be ruptured; and either from it or the gastric vessels, profuse hemorrhage into the peritoneal cavity may occur.

Should the use of the stomach tube prove unavailing in relieving the distention, prompt operation is required. If the stomach is very tense, it should be evacuated by puncture or incision; when it becomes flaccid, the puncture should be sutured, and the volvulus should then be reduced, if possible. The surgeon must remember the most frequent form of volvulus (around a transverse axis), for it is often impossible to determine by inspection how the viscera came to occupy the positions in which they are found. The transverse colon should be sought: usually it will be found close beneath the liver or diaphragm, and the spleen may be beneath the ensiform process, in the neighborhood of the gall-bladder, or even in the pelvis. The posterior wall of the stomach usually presents, and has to be tapped; then as the evacuation proceeds the site of puncture may become inaccessible, and Berg was forced to suture his first puncture before the stomach was half empty, and to make another incision in that part of the gastric wall which then became more accessible. If reduction cannot be accomplished, the stomach should be drained, to prevent subsequent distention, and in the hope that spontaneous reduction may subsequently occur. If feasible this drainage is to be procured by means of gastro-jejunostomy; if this cannot be done, a gastrostomy will suffice. When reduction has been successfully accomplished, the stomach need not be drained, and it will not usually be requisite to seek to prevent a recurrence of the volvulus by gastropexy. In Berg's first patient no recurrence was noted, and he was reported as well more than ten years after the operation. (See Borchardt, loc. infra cit.)

There are at least 22 operations for gastric volvulus on record. These are abstracted below; 7 patients died, and 15 recovered.

Operations for Volvulus of the Stomach

1. Berg, 1895. Evacuated by trocar, untwisted. Recovery.
2. Berg, 1896. Untwisted. Recovery.
3. Borchardt, 1906. Gastrotomy; stomach evacuated and incision sutured; volvulus irreducible; profuse bleeding from spleen; packed. Death.
4. Bourcart, 1913. Reduction; gastroplasty (hour-glass stomach). Recovery.
5. v. Bornsdorff, 1909. Reduction and cecostomy (for accompanying torsion of transverse colon). Recovery.

6. Delangre, 1907. Aspiration and reduction; gastropexy. Recovery.
7. Dujon, 1903. Inoperable. Death.
8. Gussander, 1911 (cited by Hedlund, 1911). Reduction, gastro-anastomosis; gastrostomy. Death.
9. v. Haberer, 1912. Pylorectomy. Recovery.
10. Hermes, 1908. Gastrostomy. Recovery.
11. Jiano, 1909. Reduction. Death.
12. Kocher, 1914. Reduction. Recovery.
13. Neumann, 1906. Reduction. Recovery.
14. Niosi, 1907. Reduction and gastro-jejunostomy. Recovery.
15. Orth, 1913. Reduction; unilateral exclusion of pylorus; jejunostomy. Recovery.
16. Bayer, 1904. Reduction. Death.
17. Rendl, 1904. Puncture and evacuation; reduction. Recovery.
18. Schüler and Walther, 1911. Laparotomy. Death.
19. Sinjuschin, 1906. Laparotomy. Death.
20. Türmoos, 1909. Reduction; gastro-jejunostomy. Recovery.
21. Werner, 1911. Reduction; gastro-enteropexy; Recovery.
22. Wiesinger, 1901. Puncture and evacuation; reduction. Recovery.

Cases of Volvulus of the Stomach

I. Around anteroposterior axis.
   (*a*) Clockwise:
      1. Kocher (Deutsch. Zeit. f. Chir., 1914, cxxvii, 591.)
   (*b*) Contra-clockwise:
      1. Streit (Amer. Jour. Med. Sc., 1906, i, 967).

II. Around transverse axis in frontal plane.
   (*a*) As an advancing wheel:
      1. Delangre (Revue de Chir., 1907, xxxvi, 603).
      2. Payer (Mitt. a.d. Grenzgeb. d. Med. u. Chir., 1909, xx, 686).
   (*b*) As a retreating wheel:
      1. Berg (Nord. med. Arkiv, Fest-Band, Stockholm, 1895, F. 11, 1; cited by Dujon: Gaz. Méd. de Paris, 1903, lxxiv, 173).
      2. Berg (Ibid., loc. cit.).
      3. v. Bonsdorff (Finska Läkaresällsk., in Jahresber. f. Chir. 1909, xv, 692).
      4. Borchardt (Arbeit. a.d. chir. Klinik (Bergmann), Berlin, 1906, xviii, 104).

5. Bourcart (Rev. de. Chir., 1913, xlviii, 800).
6. Collischonn (Beitr. z. Kasuistik d. Form u. Lagerungsstörungen d. Magens, 1888; cited by Mühlfelder: Arch. f. Verdauungskr., 1911, xvii, 53).
7. Collinschonn (Ibid., loc. cit.).
8. Dujon (Gaz. Méd. de Paris, 1903, lxxiv, 109).
9. Dupre (cited by Mühlfelder: Arch f. Verdauungskr., 1911, xvii, 53).
10. Hahn (Vortr. i.d. aerzt. Vereinig. v. Nürnberg, 1907; cited by Mühlfelder: Arch. f. Verdauungskr., 1911, xvii, 53).
11. Hedlund (Mühlfelder: Arch. f. Verdauungskr., 1911, xvii, 53).
12. Hermes (Deutsch. Zeit. f. Chir., 1908, cxv, 310).
13. Jiano (Bull. Soc. d. Sc. Méd. de Bucharest, 1909. 16 juin, p. 131).
14. Kerr (Annals of Surgery, 1912, lvi, 697).
15. Niosi (Riforma Med., 1907.).
16. Pendl (Wien. klin. Woch., 1904, xvii, 476).
17. Schüler and Walther (Arch. f. Verdauungskr., 1911, xvii, 82).
18. Sinjuschin (Chirugia, 1906; in Centralbl. f. Chir., 1907).
19. Tuffier and Jeanne (Rev. de Gynéc. et de Chir. Abdom. 1912, xviii, 27).
20. Wiesinger (Deutsch. med. Woch., 1901, xxvii, 83).
21. Wilke (Münch. med. Woch., 1907, liv. 1012).

III. Around longitudinal axis in sagittal plane.

(*a*) Clockwise when viewed from patient's head:

1. Berti (Gaz. Med. Ital. Venete, Padova 1866, ix, 139; cited by Dujon: Gaz. Méd. de Paris, 1903, lxxiv, 109).
2. Oltmann (Ein Fall v. hernienartiger Vorwölbung d. Zwerchfell m. Achsendrehung u. Zerreissung d. Magens. Kiel, 1889).

(*b*) Contra-clockwise when viewed from patient's head:

1. v. Haberer (Verhandl. d. Deutschen Gesellsch. f. Chir., 1912, xli, 197).
2. Krymholz (Chirugia, 1911, xxix, 409; in Jahresber. f. Chir., 1911, xvii. 429).
3. Langerhans (cited by Dujon: Gaz. Méd. de Paris, 1903, lxxiv, 109).

4. Mazotti (Rivista Clinica di Bologna, 1899, iv, 280; cited by Dujon: Gaz. Méd. de Paris, 1903, lxxiv, 109).
5. Neumann (Deutsch. Zeit. f. Chir., 1906, lxxxv. 136).
6. Orth (Wien. klin. Woch., 1913, xxvi, 457).
7. Saake (Virchow's Arch. f. path. Anat., 1893, cxxxiv, 181).

IV. Unknown forms.

1. Türmoos (cited by Mühlfelder: Arch. f. Verdauungskr., 1911, xvii, 53).
2. Werner (Ibid.).

FIG 63.—Eventration of the Diaphragm. Skiagram, Anterior View, with Patient Prone: A, Heart Shadow in Right Thoracic Cavity; B, Left Half of Diaphragm Rising to the Lower Margin of the Third Rib, C. (*H. G. Wood, in Surgery, Gynecology and Obstetrics.*)

Eventration of the Diaphragm.—(Eventratio Diaphragmatica.) This rare condition, first described by Petit in 1790, and which is sometimes confounded with diaphragmatic hernia, is defined by Sailer and Rhein (1905) as "an abnormally high position of the left half of the diaphragm, with dislocation upward of the abdominal viscera, particularly the stomach, on the left side; hypoplasia of the left lung, and displacement of the heart to the right." Fischer (1914) referred

to 23 cases, to which may be added others since reported by Krause (1913), Stein (1914), Upham (1914), and Wood (1916). The accompanying illustrations are from Wood's article (Figs. 63, 64).

As a rule, the **symptoms** closely resemble those of diaphragmatic hernia, which is more frequent on the left than on the right side; but there is in eventration of the diaphragm no history of sudden onset nor of trauma; in the immense majority of cases the condition is congenital, though Sailer and Rhein consider it possible that an acquired form may exist. It has been suggested that this may be due to a

FIG. 64.—Eventration of the Diaphragm. Skiagram, Posterior View, with Patient Prone, Buttocks Being Elevated and Shoulders Lowered: A, Heart Shadow in Right Thoracic Cavity; B, Left Half of Diaphragm in Contact with Cardiac Portion of Stomach, C, Which is Well Filled with Barium; and the Gas Bubbles, D, in the Pyloric Part of the Stomach. (*Wood.*)

lesion of the phrenic nerve. Although, as has been stated there is usually no history of a sudden onset, there may be exacerbations of the symptoms. These, if manifested clinically in any way, are apt to be characterized by dyspnea and cardiac palpitations. The condition is seldom accurately diagnosticated until autopsy. During life the most prominent physical signs are dextrocardia and tympany in the lower left chest. The differential diagnosis from pneumothorax

and diaphragmatic hernia is important; indeed, it is only its resemblance to the latter condition that renders it of interest surgically. In pneumothorax some cause for the anomaly usually may be discovered, dating its development; respiratory excursion is absent or limited, and the upper border of the tympanitic area does not move during respiration, whereas in eventration of the diaphragm this is a constant sign. By means of the stomach tube, distention of the stomach with air or liquid will demonstrate its position, and a skiagraphic examination may show that the diaphragm retains its normal level in pneumothorax, whereas in eventration of the diaphragm it is markedly elevated. Diaphragmatic hernia usually can be excluded by the history. In one case of eventration of the diaphragm studied by Widemann (1901), a diagnosis of diaphragmatic hernia was made a year later by Glaser. The patient, 48 years of age, had had four attacks of pleurisy. Profuse gastric hemorrhage ushered in the last attack, and the patient was then taken to the hospital. All the signs of diaphragmatic hernia were present, but the first skiagraph made showed that the left side of the diaphragm was abnormally high, and that the stomach was not above it (Widemann.) "Later a transverse line below the clear area could be seen moving with the respiratory excursion, and a diagnosis was made of diaphragmatic hernia." (Glaser, quoted by Sailer and Rhein.) As the hematemesis was repeated, the hernia was thought to be incarcerated. At the operation (done by Körte) no hernia was found. The patient recovered, but died a year later from carcinoma of the tongue.

The **treatment** of patients with eventration of the diaphragm can only be prophylactic of complications, though Wood (1916) suggests that plication of the diaphragm might prove of benefit.

Diaphragmatic Hernia.—Diaphragmatic hernia is not very rare. Salomoni in 1910 was able to find records of about 1000 cases. This form of hernia may be classified as congenital and acquired; but here, as in other hernias, a distinction must be made between hernias which are present at birth, and those which, though due to some congenital defect in the diaphragm, do not develop until some time after birth, occasionally not until late in life. Either form of hernia, moreover, may happen to be Intrapleural, Subpleural, Subdiaphragmatic, or even Intrapericardial as in the case recorded by Grenier de Cardenal (1903). Probably the least usual form is the subdiaphragmatic, which is also known as "true diaphragmatic hernia." In this variety the diaphragm becomes pouched, and the muscular fibres, even if weak and indistinct, may nevertheless be traced throughout the walls of

the hernial sac. If the protrusion occurs through a defect in the diaphragm, whether congenital or acquired (by stabwound, gunshot wound, or traumatic rupture of the diaphragm), the sac may still be lined by peritoneum; but this is very unusual. The pleura also is usually absent over the hernia, the variety most frequently encountered being that in which the herniated viscera (stomach, colon, etc.) lie free in the pleural cavity—intrapleural hernia.

The hernia is much more often on the left than on the right side. Among 282 cases studied by Deitz (1884), in 261 (93.5 per cent.) the hernia was on the left. The reason for the immunity of the right side has always been said to be the presence of the liver beneath the diaphragmatic vault, acting as a buffer, and materially strengthening the right half of the diaphragm. There are certain regions of the diaphragm in which hernia is more apt to occur than in others; these are not, as might be expected, the natural phrenic openings, such as the esophageal and caval orifices. Hernia through these is rare. There is, however, a triangular area found between the costal and sternal attachments of the diaphragm, which is filled in only with areolar tissue, and where the abdominal and thoracic cavities are not separated by muscular fibres. This weak spot is sometimes the seat of a hernia; but more often the protrusion occurs in the neighborhood of the left leaflet of the central tendon of the diaphragm. In congenital hernia the defect is usually in the posterior half of the diaphragm, which is a later development than the ingrowth from the ventral surface of the fetus which forms the anterior segment of the diaphragm. In other cases the hernia is situated in the posterior part of the diaphragm, along the outer margin of the left crus, and beneath the internal arcuate ligament. The most frequently herniated viscera, according to Deitz, are the stomach, colon, omentum, small intestine, spleen, liver, duodenum, cecum, and kidney—in the order named. The bladder, the rectum, and the female generative organs have never been found in a diaphragmatic hernia.

Most cases of diaphragmatic hernia are observed in the fetus, or in infants stillborn, or dying very soon after birth. In Lacher's (1880) cases, the age was recorded in 204 instances, and of these no less than 80 (40 per cent.) were in infants less than one year old, or in the fetus. A child so malformed from birth is very badly equipped for the struggle for existence, and is prone to succumb to intercurrent maladies, especially pulmonary affections. Sudden death from acute cardiac incompetency is a frequent termination, especially in adults. Indeed it has been said that diaphragmatic hernia should always be considered in deciding the cause of a sudden death.

Males are supposed to be more subject to this affection than are females, especially men in occupations which subject them to injuries or to sudden violent strains. Most cases seen in adults are due to *previous injury of the diaphragm* (rupture, stab or gunshot wound).

Subjective **symptoms** are often wanting, the malformation being unexpectedly found at autopsy. In the newborn, cyanosis and dyspnea are prominent; the left thorax does not expand normally; there is dextrocardia; and death usually ocurs within a few hours. The adult patient may have suffered from mild indigestion, with borborygmi, and tympany after eating; and this condition may have continued for years without material discomfort; at any time, however, acute overdistention of the herniated stomach may cause sudden cardiac failure, perhaps death; or strangulation of the hernia may arise from a strain which forces a larger portion of the abdominal contents through the diaphragmatic opening. Great thirst is a symptom on which stress is laid by many writers. The symptoms due to stabwounds or gunshot injuries of the diaphragm, with protrusion of the stomach or colon, are usually overshadowed by those due to the injuries to the abdominal viscera involved.

The **physical signs** of a diaphragmatic hernia are much more precise in theory than in practice. We know certain so-called pathognomonic signs, by means of which diaphragmatic hernia may be distinguished from pneumothorax and other conditions which it resembles more or less closely; but when practical application is made of the tests, it must be acknowledged that both physician and surgeon frequently remain undecided as to the true condition present. Among the 276 cases collected by Lacher in 1880, only seven were diagnosticated during life; and though our diagnostic acumen has greatly increased, in regard to abdominal diseases, during the last generation, still it must be confessed that even yet the diagnosis of diaphragmatic hernia may be difficult. Giffin (1912) found 15 cases in which the true condition was recognized during life, without, or before operation. We have references to 7 subsequent case reports in which the diagnosis was made clinically or by the aid of the X-rays. The physical signs and tests employed are fully described in most textbooks on the practice of medicine, and need be outlined here very briefly: The lower chest on the affected side is tympanitic; the breath sounds are absent or very feeble and distant; vocal fremitus is lost; expansion is decreased; and the heart is dislocated away from the affected side—that is to say, there is usually dextrocardia. The same signs exist in pneumothorax;

but in diaphragmatic hernia the diaphragm does not descend on deep inspiration, and causes which may produce pneumothorax may nearly always be absolutely excluded, while a history of sudden onset following severe strain (sometimes childbirth) or a crushing injury, or occurring some years after a stabwound or gunshot wound of the thorax, is highly characteristic of diaphragmatic hernia. Inquiry as to trauma some months or even years previously is important, as in several cases such a history has revealed the predisposing cause of the hernia, and thus confirmed a diagnosis tentatively made from the symptoms alone. In such cases the defect in the diaphragm may be congenital, and the previous accident may have been the first cause of prolapse of abdominal viscera through the opening; but no symptoms may have been noted until the recent strain, which produced incarceration or strangulation.

Moreover, introduction of a stomach tube and distention of the stomach with air or liquid (preferably the latter) will very quickly change the physical signs in the case of diaphragmatic hernia, while in pneumothorax the thoracic tympany and other signs will not be affected. The succussion splash, so characteristic of pneumothorax, may also be elicited with great clearness in many cases of diaphragmatic hernia; but filling the stomach with fluid will have no effect on this phenomenon if due to pneumothorax, while it will be speedily abolished, if it was caused by fluid in an air-containing stomach. Aspiration is to be condemned as a method of diagnosis, the dangers of consequent septic pleuritis or peritonitis being very great. The distinction between eventration of the diaphragm and diaphragmatic hernia has been referred to at page 235.

A further aid in the diagnosis of diaphragmatic hernia is the Roentgen ray. The level of the diaphragm may thus be detected; and by introducing bismuth emulsion or a stomach tube filled with mercury into the gastric cavity, its relation to the diaphragm may usually be determined.

Treatment.—There is no question that in cases of diaphragmatic hernia suddenly developed, and of evident traumatic origin, immediate reduction by operative means offers the greatest prospect of recovery. In such cases, as in other irreducible hernias so acquired, the danger of strangulation is particularly great, and the injury may have produced lesions of the herniated organs (rupture, hemorrhage, etc.) which can be treated safely only by surgical means. According to Lenormant (1903), among 33 cases of wounds of the diaphragm in which no operation was done, collected in 1893 by von Frey, there were 29 deaths; and

among 21 unoperated cases analyzed in 1901 by de Font-Reaulx, there were 16 deaths, and 3 cases in which diaphragmatic hernia subsequently developed. On the other hand, Lenormant himself collected records of 31 patients with wounds of the diaphragm which were operated upon, and of these only 7 died. Although this comparison refers only to stab-wounds and gunshot injuries, the conditions in subcutaneous rupture and in cases of diaphragmatic hernia of sudden development are so similar that the figures given are applicable also to injuries in which no external wound exists. In cases where it seems probable that the hernia is of long duration, and due to a congenital defect in the diaphragm, delay in resorting to operation is justifiable; but if the signs of incarceration or obstruction arise, no further time should be lost—the hernia must be reduced by operative means before strangulation has made reduction useless by producing edema, sloughing, and gangrene of the herniated viscers.

According to John Wood (1888) it was Guthrie who first proposed "to make an opening in the abdominal cavity, and to introduce the hand in order to withdraw the bowel from the hernial opening." Permann and Postempski proposed in 1889, independently of each other, the operation of thoracotomy, Permann advocating it for ordinary cases of diaphragmatic hernia, while Postempski urged it, and successfully employed it in several cases, for wounds of the diaphragm. The first operation (laparotomy) in a non-traumatic case appears to have been carried out in 1879 by Bardenheuer (the hernia not being discovered until postmortem examination), while Postempski in 1889 did the first operation (thoracotomy) for stab-wound. Naumann, in 1888, appears to have been the first to find the hernia at operation (laparotomy); he was, however, unable to reduce it. Surgeons are divided as to the route by which the rent in the diaphragm should be approached, some preferring laparotomy, but most are in favor of thoracotomy. In 1910, Salomoni collected 51 operations, with 39 deaths (76.2 per cent.): 11 thoracotomies, with 4 deaths (36.3 per cent.) and 39 laparotomies with 35 deaths (89.7 per cent.). In most if not all of the early cases, operation was done only when strangulation occurred, and in most of these the only pre-operative diagnosis was intestinal obstruction, and in many the cause of the obstruction (diaphragmatic hernia) was not found at operation (laparotomy).

In cases where strangulation is not present, and where the true condition is recognized before operation, or is found accidentally during operation for some other condition, the prospects of recovery

are excellent. Unless the patient's condition forbids it, we believe that in such cases reduction of the hernia and repair of the diaphragmatic opening should be attempted. Among 11 recent cases of this kind to which we have reference (Beckmann, 1909 (3 cases); Bythell (1915); Carson (1912); Cranwell (1908); Giffin (1912); Harris and Greene (1912); McGuire (1914), 2 cases; Scudder (1912)), there were no deaths but in several of these cases no attempts were made to repair the diaphragm. Four recent operations for strangulated diaphragmatic hernia (Bamberger, 1910; Caffee, 1911; Litler-Jones, 1909; Thompson, 1909) are on record, with only 2 recoveries.

In favor of thoracotomy is the fact that the lung is already collapsed and the heart displaced, so that even wide opening of the pleural cavity could not materially increase the danger on that score; the greater accessibility of the diaphragmatic opening also would make us prefer thoracotomy. More important, however, than either of these factors, is the existence of negative pressure in the unopened pleura; the herniated viscera are thus held in the pleural cavity by suction, and reduction by traction from within the abdomen is difficult, if not impossible. Indeed, the only reason we can see for preferring the abdominal route would be the possibility of injury to some abdominal organs which could not be repaired from above. A study of the cases, however, in which operation has been done for diaphragmatic hernia, has convinced us that *in the immense majority of cases thoracotomy should be the operation employed.* Unless local anesthesia is employed, the anesthetic (ether) should be administered by intratracheal insufflation. The technique consists in opening the pleural cavity, reducing the hernia, repairing the defect in the diaphragm, and closing the primary incision. In many cases an intercostal incision, preferably in the eighth interspace, will give sufficient exposure if the ribs are forcibly drawn asunder by strong retractors. The flap operations of Postempski, Rydygier, and others are not necessary, and should therefore not be employed. If the simple intercostal incision does not give sufficient exposure, one or two ribs—those bordering on the primary incision—may be resected for a distance of four or five inches. More room than is thus obtained is seldom requisite. Should temporary resection of the chest wall be employed, Rydygier's operation is to be preferred to that of Postempski. Rydygier made an intercostal incision, enlarging the existing stabwound, and joined this at its posterior extremity by an incision made downward from it, practically at right angles to the ribs. The ribs are then divided in the line of the second incsion, and the osteo-

plastic flap thus formed is turned downward and forward, the elastic costal cartilages, which are not divided, acting as a hinge. In Postempski's operation the ribs are divided front and back, and the flap thus made is turned directly downward. In Cranwell's operation (1908) a flap similar to Postempski's was employed, but with the base above. In some instances the flap formed by Postempski's method has sloughed. The use of hot moist compresses of silk or of handkerchief gauze, to isolate the operative field from the upper portion of the pleural cavity and the lung, is of great assistance, and lessens any symptoms which may arise from the operative pneumothorax. In cases of stabwound, or of traumatic hernia, great care should be taken to repair any injury of the stomach, colon, or other structures found in the pleural cavity. As such injuries are in the upper or posterior walls of the stomach, they are readily accessible by the transpleural route, but are very difficult of access or totally inaccessible by laparotomy. After all ruptures or perforations are repaired, the herniated organs are to be reduced, and the breach in the diaphragm sutured. If this be very large, the omentum may be attached to its margins, by sutures; but usually it has been possible to close it without the use of omentum. It is better not to drain the pleural cavity; even if, in a few cases, immediate closure of the thoracic wound without drainage results in subcutaneous emphysema, or in the development of hemothorax or empyema. These latter complications may be remedied by a secondary thoracotomy; if primary drainage is instituted in every case, the total number of cases of pyothorax will be much increased. In Riegner's patient with stab wound of the diaphragm, the abdomen was opened after repairing the herniated organs and the diaphragm by thoracotomy; but as no abdominal lesion was found, the laparotomy wound was immediately closed. While it is certainly safer to explore the abdomen if there is a probability of further lesions, in most cases such good exposure has been obtained by means of thoracotomy that no secondary laparotomy has been employed.

If no diagnosis other than intestinal obstruction has been made, laparotomy will be the operation employed; but if reduction of the hernia prove difficult from below, the surgeon should not hesitate to create a pneumothorax by means of thoracotomy, as has been done by Dennis (1905) and others, to relieve the negative pressure within the pleural cavity.

CHAPTER XI

CARCINOMA OF THE STOMACH

Carcinoma of the stomach is a disease of frequent occurrence. From 25 to 40 per cent. of all cancers in the body are primary in the stomach. Statistics show that over 13 per cent. of deaths due to diseases of the digestive organs are caused by carcinoma of the stomach. In America as well as in other civilized countries the frequency of carcinoma is increasing. According to Hoffman (1914) the number dying from cancer is about 25 per cent. greater than ten years ago. The United States Census report for 1900 recorded 9000 deaths from cancer during that year; 31 per cent. of fatal cancers were in the stomach; probably (as we shall see later) many cancers of the liver and the abdomen were primarily gastric; and many cases recorded as gastritis and allied diseases were really carcinomatous in nature (Dowd, 1906).

Etiology and Pathology.— The cause of cancer is unknown. Direct curative **treatment** is therefore at present impossible. The only way to keep the patient so afflicted from being killed by the disease is to remove it *in toto*.

In studying carcinoma of the stomach it is especially the predisposing causes of its existence that must be considered, because little is known of the exciting causes. It is shown under the discussion of Treatment that surgery is dealing effectively with some of these predisposing causes.

Race.—Carcinoma, whether of the stomach or other region of the body is peculiarly an **affection of the Caucasian race.** Friedenwald (1914), practising in Baltimore, Md., which has a large colored population, observed carcinoma of the stomach only in 52 negroes among a total of 1000 patients with the disease, or in 5.2 per cent. The yellow races seem more vulnerable than the black, while the white races are most liable to its presence.

Sex has very little influence on the occurrence of the disease. Among 1303 cases, Fox observed 680 men and 625 women; in 2214 cases studied by Welch, 1233 were males and 981 females. Fenwick (1902), after quoting these figures, gives the results of his own researches: among 3670 post mortem examinations of gastric cancer, 2162 were males, and 1517 females, a proportion of rather less than 6 to 4. But,

as Fenwick points out, it is to be remembered that men form a larger proportion of hospital patients and of the subjects of post mortem examination than do women, so that even these figures may exaggerate the relative frequency of gastric carcinoma in men. But in the statistics of Smithies (1916) recently published, among 921 patients, 693 were male, and only 228 female, a proportion of more than 3 to 1.

Age.—About 80 per cent. of all cases of gastric carcinoma occur in patients between the ages of 49 and 70 years. It is rare below the age of 20 and over that of 80 years. Bernouilli (1907) reported three cases that came under his own observation in which there was carcinoma of the stomach or rectum in patients aged 15, 17, and 18 years respectively. He also collected 50 cases from the literature in which carcinoma had been found in the young, the stomach being the site of disease in 13. In one instance the cancer of the stomach was evidently congenital.

Trauma may determine the occurrence of carcinoma in the stomach. Ropke (1905) found trauma a direct cause of gastric carcinoma in 2 out of 79 cases observed; Friedenwald (1914) obtained a definite history of trauma in 19 out of 1000 patients, nearly 2 per cent.; while Smithies (1916) reports it as a cause in 2.6 per cent. of his 921 cases. Lacerations of the mucosa, from acute distention of the stomach, have been suggested by Strassmann (1907) and others as the starting point of gastric ulcers and cancers. (See p. 298.)

Simple Gastric Ulcer.—There is no factor which of late years has attracted so much attention as a predisposing cause of gastric cancer as has simple gastric ulcer. Long recognized under the terms "chronic gastritis," "dyspepsia," "indigestion," etc., a more intimate knowledge of these cases has shown that in most patients these digestive disturbances were due to the presence of an ulcer or its sequels (cicatrices, stenosis of the pylorus, etc.). And the evidence that carcinoma is frequently an end result of such lesion has been accumulating for more than a decade. Thus Mumford and Stone (1905) traced 60 patients who had been treated at the Massachusetts General Hospital for "chronic indigestion," and who subsequently died. Of these no less than half died of gastric carcinoma. These writers further made a study of 50 patients with gastric carcinoma and learned that in no less than 41 of the 50 patients there was "a history either of ulcer or of long-continued digestive disturbance, of which the exact nature could not be ascertained." W. J. Mayo in 1905 found a history of ulcer or other gastric disease in 36 per cent. of his patients with cancer of the stomach, and detected clear evidence of cancer having developed on ulcer in 30 per cent. of the last forty partial gastrectomies performed; in 1907 he

reported that the clinical history of 69 patients with gastric cancer, and pathological examination of the specimens removed from them by gastrectomy, during 1905 and 1906, made it clear that in 54 per cent. the cancer had its origin in an ulcer. In 1914 MacCarty and Broders reported, from the Mayo Clinic, that 472, or 69 per cent., of 684 specimens of gastric lesions, excised or resected at operation, presented characteristics of simple ulcer *plus* carcinoma; in 3 per cent. more the existence of carcinoma was doubtful; while only in 191 (28 per cent. of the total) was there certainly no carcinoma. Of 399 operation speci-

FIG. 65.—Microphotograph (Low Power) showing Carcinoma Beginning in Edge of Gastric Ulcer. See Fig. 66. From a Patient in the Lankenau Hospital. (*Dr. Reiman.*)

mens of gastric carcinoma studied by Wilson and McDowell (Mayo Clinic, 1914):

53.6 per cent. showed unmistakable evidence of previous ulcer,
4.8 per cent. showed doubtful evidence of previous ulcer, and
42.6 per cent. showed no evidence of previous ulcer.

Kocher (1912) found in his series only 8 to 9 per cent. with microscopical evidence of preceding ulcer, but in 72 per cent. of his patients with cancer of the stomach there was a history of ulcer; and from 13 to 43 per cent. of lesions excised under the diagnosis of ulcer proved really to be carcinomatous. Our own statistics do not give so high a percentage: of 143 patients operated on at the Lankenau Hospital for gastric carcinoma (1909 to 1920), 44, or 30.7 per cent., gave a previous history

characteristic of ulcer; and among the last 100 specimens of gastric carcinoma removed at operation, which have been carefully studied by Reiman (1920) 38 gave evidence of having developed on an ulcer base. (Figs. 65 and 66.) Physicians claim a much lower incidence of previous ulcer history than most of the figures quoted above. Thus Friedenwald, among 1000 cases of carcinoma of the stomach, noted a history of previous digestive disturbance only in 23 per cent., and only 7.3 per cent. gave a definite history of ulcer. Lockwood (1913) notes that of 174 cases of cancer of the stomach in which the previous history was complete, 148

FIG. 66.—Microphotograph (High Power), showing Early Carcinoma (see Fig. 65).

(86 per cent.) gave no history of previous digestive trouble; 13 (7 per cent.) had an indefinite history of indigestion, not suggesting ulcer, and only 13 in all (7 per cent.) gave a history that indicated previous ulceration. Smithies reports the following figures in a study of 921 cases of carcinoma of the stomach:

Ulcer, with microscopical evidence of carcinoma, 7.8 per cent.; average duration of symptoms 13.7 years.

Clearly cancer when first seen, with a history of ulcer, 47.3 per cent.; average duration of ulcer symptoms 10.5 years, succeeded by average period of malignant symptoms for 6.3 months.

No previous stomach history, 31 per cent.; average duration 7 months.

Previous vague stomach history, 9 per cent.; average duration 9 years, with an average period of malignant symptoms for 6.2 mos.

Few clinical signs of stomach disease, 2 per cent.

Carcinoma of stomach secondary to carcinoma elsewhere, 1.7 per cent.

Almost alone among modern surgeons Paterson (1914) argues against the frequency of gastric ulcer as a previous lesion to gastric carcinoma. Küttner (1914) also appears to hold this view, as the result of his study of material from 1100 operations; but he claims that a large number of callous ulcers of the stomach are malignant from the very beginning. If Paterson's arguments are carefully analyzed, it will be found that he is reduced to the same conclusion. And it is interesting to note a statement by Judd of the Mayo Clinic (1919) that it is now generally recognized that most carcinomata of the stomach have not originated in simple ulcers but have been malignant from their beginning.

It should be noted also that other pathologists than those associated with the Mayo Clinic have not accepted as evidence of carcinomatous transformation in ulcers changes which Wilson, MacCarty and their colleagues have exhibited as such.

Konjetzni (1913) collected statistics on this subject from various clinics, comprising a total of 2337 cases of carcinoma of the stomach: the number which were thought, after histological study, to have originated as simple ulcers varied from 1 to 22 per cent.; so that he regarded Wilson and MacCarty's figures (71 per cent.) as altogether exceptional.

A sensible conclusion, it seems to us, is that in from one third to one half of all gastric cancers the history is that of ulcer, and that *early diagnosis and treatment of gastric carcinoma consists in diagnosis and operation on cases of gastric ulcer.*

The **histological changes** by which simple ulcer becomes converted into carcinoma have been particularly investigated by the French pathologists, and in this country especially by MacCarty and Wilson, of the Mayo Clinic. Although, as already indicated, their theories are not unreservedly accepted by other writers, they seem to merit a short description in this place, especially as, when considered in this light, these changes may be regarded as in the nature of predisposing causes. Hayem described, as long ago as 1895, certain adenomatous changes in the pyloric region, which he characterized as Brunnerian in

type—that is to say, resembling the glands of Brunner, which are found in the duodenum, and which are distinguished from the gastric glands by their situation within the submucosa, the gastric glands, as is well known, never extending below the muscularis mucosæ. These adenomata of Brunnerian type, occurring in the pyloric region of the stomach, are clearly neoplastic in character, and were regarded by Hayem as an early stage of carcinomatous transformation. Ménétrier in 1900 elaborated earlier studies by himself along the same line. He summed up his theory of carcinomatous transformation in this way: First stage—this is purely inflammatory; there is a chronic gastritis and the cells lining the glands lose their special and distinctive features (the histological picture is simplified); and the "acid" cells disappear. Second stage—adenomatous in character; the proliferating glands, deprived of their characteristic elements, become more contorted and convoluted; the cells increase in number; cysts form as the result of obstruction of the gland ducts by proliferation of their lining cells. Third stage—epitheliomatous in character; the cells break through the muscularis mucosæ and finally are found lying free among the connective tissues of the gastric walls. It is the great frequency with which MacCarty found similar changes in the periphery of gastric ulcers, rather than anything new histologically, that renders his investigations of interest. It must be acknowledged that the statement of Ewing, to the effect that a histological picture does not have to be either malignant or non-malignant, but that it may be neither the one nor the other, represents a very convenient state of mind for pathologists to maintain.

Clinical Pathology. Situation.—The following table shows the location of the growth in 1850 cases collected by Fenwick, as well as in 854 cases studied personally by Smithies:

Distribution of carcinomata in the stomach	Pylorus	Lesser curvature	Cardia	Posterior wall	Greater curvature	Anterior wall	Fundus	Whole or greater part	Multiple growths
Fenwick (per cent.)....	58	11.5	9.8	5.0	2.8	2.2	1.5	6	2.9
Smithies (per cent.)....	42	24.7	3.5	9.3	2.3	2.3	0.9	12	2.9

Fenwick concluded from his figures, "that in 79.4 per cent., or in about four-fifths of all cases, carcinoma commences in the comparatively small strip of tissue which extends from one orifice to the other along the upper margin of the stomach, and that its percentage rapidly

diminishes the further we proceed from the pyloric valve." The nearly identical distribution of cases of callous ulcer is a further indication of the probable relation between the two affections.

Histological Structure.—Microscopically, three types of gastric cancer are recognized: (1) A tumor composed of spheroidal cells like those normally lining the gastric tubules (spheroidal celled carcinoma); (2) a tumor composed of more or less columnar or cylindrical cells, similar to those normally lining the pyloric glands (cylindrical celled or adeno-carcinoma); (3) a tumor whose chief characteristic is myxoma-

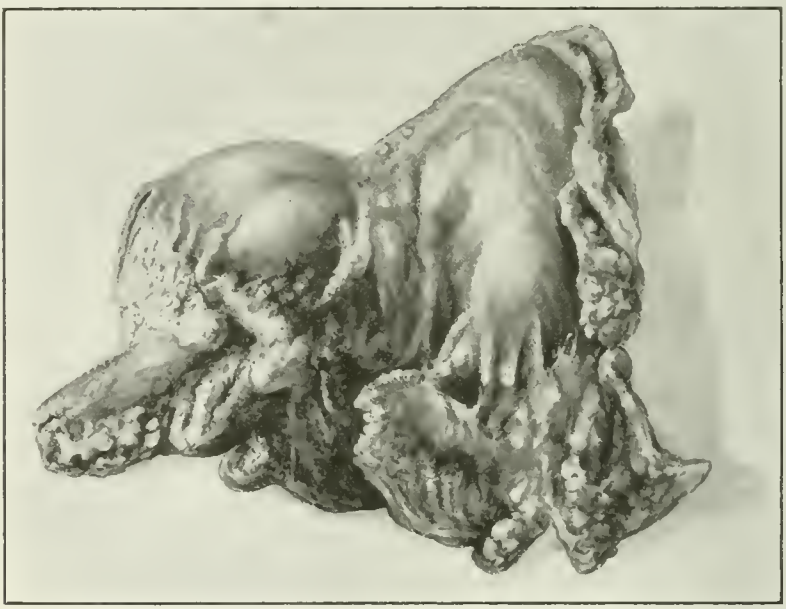

FIG. 67.—Carcinoma of Stomach; Specimen Removed by Partial Gastrectomy. Man 43 Years of Age, Gastric Symptoms for Two Months Only; Loss of Weight 52 lbs. Tumor Doubtfully Palpable. In Good Health, Free from Symptoms, and has Gained 40 lbs. in Weight, 16 Months after Operation. See Fig. 68. From a Patient under the Junior Author's Care in the Episcopal Hospital.

tous degeneration of epithelial cells and stroma (colloid carcinoma), which may be the result of changes either in the spheroidal celled or the cylindrical celled carcinoma. Finally these tumors are described as scirrhus or medullary (encephaloid) according as they are rich or not in fibrous tissue as compared with the cellular elements present. It is perhaps well, from a clinical point of view, to bear in mind also the term (4) *ulcus carcinomatosum*, or carcinomatous ulcer (Fig. 68), which indicates that the main feature of the growth is an ulcer, whether the ulcer was originally benign or was malignant *ab initio*, or was the result of sloughing of a scirrhus or a medullary carcinoma.

There does not appear to be sufficient material for it to be decided which variety of carcinoma occurs most frequently. As a matter of fact, the spheroidal and the cylindrical cell types both may be present in the same tumor, or colloid changes may completely obscure the field. Brinton's figures (1857) indicated that 72 per cent. of gastric carcinomata were of the scirrhous variety; but more recent statistics compiled according to Brinton's method tend to show that the medullary forms

FIG. 68.—Carcinoma of Stomach: Specimen Shown in Fig. 67 has been Opened Along the Lesser Curvature; Exposing Large Callous Ulcerated Cancer Involving Nearly Entire Circumference of Stomach. From a Patient under the Care of the Junior Author in the Episcopal Hospital.

predominate. Fenwick studied 115 cases of gastric carcinoma microscopically, and reported 63.5 per cent. as spheroidal celled, 28.6 per cent. as cylindrical celled, and 7.8 per cent. as exhibiting signs of colloid degeneration.

Metastasis occurs early in carcinoma of the stomach, but for a reasonable time this metastasis is confined to the immediately adjacent lymph nodes. According to Mumford, in from 4 to 10 per cent. of those patients with the perigastric lymph nodes palpably enlarged, no

carcinomatous invasion of these lymph nodes exists. The lymphatics of the stomach have already been discussed (p. 13). Our knowledge of these lymphatic areas is due almost entirely to the classical investigations of Cunéo (1903); and Hartmann (1904) was the first to make practical applications of his teachings in performing excision for gastric cancer. More recent investigations have invalidated the conclusions of Cunéo in some anatomical details, but the practical lessons to be drawn from Cunéo's researches are in no way affected. The chief of these lessons is that carcinoma, beginning, as it usually does, along the lesser curvature close to the pylorus, invades first the lymph nodes

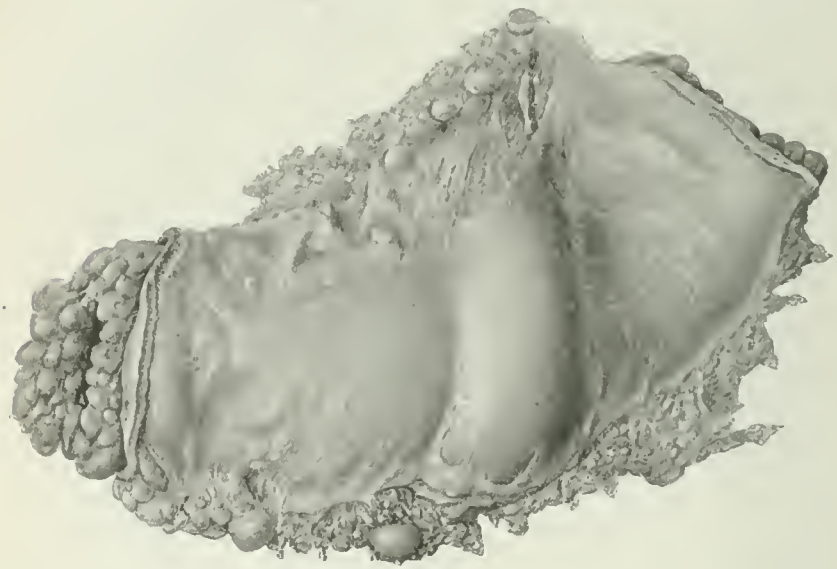

Fig. 69.—Carcinoma of Stomach: Specimen Removed by Subtotal Gastrectomy, see Fig. 70. From a Patient under the Senior Author's Care in the Lankenau Hospital.

lying along the lesser curvature; and that this chain of lymph nodes is very quickly invaded even up to the coronary group of nodes surrounding the coronary artery close to the cardiac orifice. From this fact it is evident that *radical operations for gastric cancer must remove practically the entire lesser curvature of the stomach.* Moreover, as soon as the carcinoma is at all extensive, it is found that the lymph nodes in the gastro-colic omentum, for a variable distance away from the pylorus, are involved. Hence *Hartmann's line* for gastrectomy was made to pass from the coronary artery to a point nearly directly below it, on the greater curvature of the stomach (Fig. 111). A third point of the greatest importance is that whereas the carcinomatous invasion

extends rapidly and for an indefinite distance away from the pyloric region of the stomach, it invades the duodenum only rarely. The removal of the first 2 cm. of the duodenum will nearly invariably enable the surgeon to get safely beyond the limits of the malignant growth. It is a well-recognized fact that the palpable induration of the gastric cancer stops with the area of mucosa affected, but that in the submucosa the invasion will have advanced considerably further: hence the necessity of cutting wide of the indurated margins of the carcinoma. From Borrmann's studies of resected stomachs from the clinique of

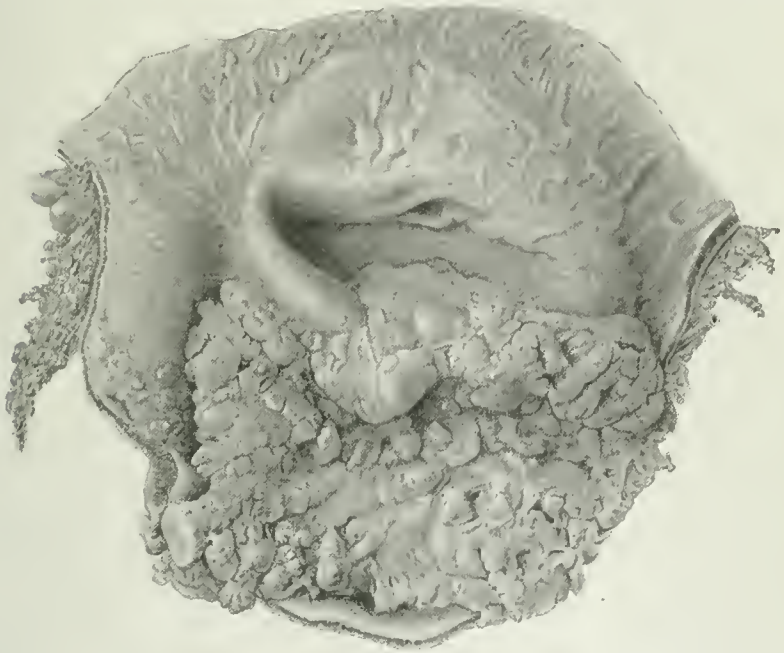

FIG. 70.—Carcinoma of Stomach: Looking Toward the Pylorus. Specimen Shown in Fig. 69 has been Opened Part Way Along the Greater Curvature from the Cardiac End of the Specimen. (*Lankenau Hospital.*)

Mikulicz it is evident that these incisions must be made from 5 to 8 cm. (two to three and a half inches) away from the macroscopical tumor on the cardiac side of the growth, and from 1.5 to 2 cm. (one-half to three-fourths of an inch) from it on the intestinal side.

While Cunéo found that the presence of lymph nodes beneath the pylorus was very unusual, Jamieson and Dobson (1907) found them quite frequently present, thus confirming the observations of Lengemann, who noted their presence in 60 per cent. of the stomachs examined. But Cunéo's conclusion that the removal of these nodes is rarely necessary, is paralleled by Jamieson's and Dobson's asser-

tion that their removal would be extremely difficult, if not impossible, and that it probably is very rarely accomplished. And, while Cunéo thought that the pylorus and the whole of the lesser curvature drained into the lower coronary group of nodes as they are named by Jamieson and Dobson, these writers found that in not a few instances lymph channels may be traced which pass directly past these nodes and empty into the right supra-pancreatic nodes lying along the trunk of the hepatic artery. The disheartening conclusion is reached by Jamieson and Dobson, as a result of their studies, that "except as a mere matter of chance no operation for gastric carcinoma can be a radical one when once malignant emboli have commenced to reach the lymphatic glands. The only reason," they add, "for removing as many of the diseased glands as possible, is the hope that once the primary growth and the majority of the glands have been removed, the remaining glands may be able to deal with, and, perhaps, destroy, the malignant elements they contain; of this process, however, we know little or nothing."

Apart from the lymph nodes, metastasis of gastric carcinoma occurs most frequently to the liver, which is affected in one-third of cases examined at autopsy. The malignant invasion occurs along the radicles of the portal vein. In scirrhous carcinoma, and in all forms which cause marked pyloric stenosis, invasion of the liver is unusual. The great omentum becomes invaded by cancerous nodules almost as frequently as the liver, but ascites is a rather unusual accompaniment. The lungs, the intestines, and other internal organs are as a rule invaded only very late in the disease. The left supraclavicular lymph nodes are sometimes affected in the last stages of gastric carcinoma, but it is worthy of note that these nodes frequently have been found enlarged, without being affected by any cancerous change which could be detected by microscopical examination. Smithies found metastasis to these nodes in 11.5 per cent., and to the rectovesical pouch in 16 per cent. of his cases; in all these instances the condition was inoperable.

Extension by Contiguity.—Gastric carcinoma is the most frequent cause of internal gastric fistula. Of 66 cases referred to by Lieblein and Hilgenreiner, in which a gastro-colic fistula was due to disease of the stomach, it was caused by carcinoma in 47. The gall-bladder is much less often involved. In any case, it is not very unusual for a fistula thus formed to close again spontaneously before death, by the development of further perigastric adhesions. The pancreas, the liver, and, very rarely, the spleen, may be invaded

Carcinoma of Stomach. Specimen Secured by Partial Gastrectomy; the Ulcerated Area can be Seen where the Cardiac End of the Specimen has Been Turned Back like a Cuff. Note also the Enlarged Lymphnodes beneath the Pylorus. (*Albright.*) Path. No. 9251. *Lankenau Hospital.*

Face p. 254

by direct extension of the growth. Perforation of the diaphragm, and even the formation of a gastric cutaneous fistula, is sometimes observed. Fenwick refers to 22 instances of this last condition, 3 of which came under his own observation at autopsy. According to Lieblein and Hilgenreiner (1905), cancer is a more frequent cause of gastric cutaneous fistula than is gastric ulcer. They collected 26 cases due to the former, and found only 17 caused by ulcer. The reader is referred to this valuable monograph for further statistics of gastric fistulæ. (See also p. 398.)

Perforation of gastric cancer into the free peritoneal cavity is very rare, existing in only 3 per cent. of the fatal cases studied by Fenwick. Sometimes a subacute perforation occurs, with the formation of a perigastric abscess; and this, by subsequent rupture, may cause death from peritonitis. Such a case, recorded by the junior author, has already been described in connection with Hourglass Stomach. (See p. 180.)

Secondary gastric carconoma is of little surgical interest. It is found in 6 or 7 per cent. of autopsies on patients with gastric cancer (Hale White; Fenwick), and is usually (73.6 per cent. according to Fenwick) due to direct extension from some neighboring organ, such as the pancreas, transverse colon, gall-bladder, uterus (through omentum), esophagus, etc.; less frequently (21 pr cent.) it is secondary to cancer of the tongue, mouth, pharynx, upper esophagus, etc., being then perhaps due, as suggested by Klebs, "to the detachment of particles of growth, which are swallowed, and subsequently become engrafted upon the gastric mucous membrane." (Fenwick.) Engelhorn (1097) called attention to gastric carcinoma occurring simultaneously with, or secondary to, carcinoma of the ovary. He has studied 13 cases from Döderlein's clinique and suggests the propriety of examining the patient for gastric carcinoma whenever malignant disease exists in the ovary. True metastases (from mammary gland, testicle, uterus, kidney, etc.) were found in 5 per cent. of Fenwick's cases, and are usually accompanied by metastatic invasion of the lungs, liver, etc. Hence surgical treatment is rarely required in secondary carcinoma of the stomach.

One patient who was operated on by the senior author by partial gastrectomy for carcinoma, returned 30 months later with a large pelvic tumor. At the operation this was found to be a solid tumor of the ovary; it was thought to be carcinomatous; but after microscopical study Dr. A. O. J. Kelly pronounced it a *sarcoma*. There was no recurrence of carcinoma in the stomach or elsewhere.

Symptoms.—Cancer of the stomach presents clinically two forms. In one, a patient past middle life, without having suffered previously from indigestion, suddenly loses appetite, especially for meats, grows progressively weaker and more emaciated, develops epigastric pain and possibly a palpable mass, becomes subject to vomiting spells every few days, which bring up a quantity of coffee-ground material, foul smelling and fermented, and quickly develops the cancerous cachexia. This is the classical picture of gastric carcinoma and it is still seen; but it is of rarity when compared with the other course of development which we have begun to appreciate only in the last decade. In the group of cases studied by Smithies this rapid onset of the disease was observed in less than a third of the whole number of patients, the average duration of the symptoms, before the patients came under observation, being 7 months.

The second, and much more frequent course, is found in patients who have been life-long sufferers from dyspepsia. Medical treatment has at times given relief, but the indigestion recurs again and again. Appetite may be preserved, but digestion is torture; so abstemiousness becomes second nature. There may or there may not have been some period when gastric ulcer was suspected or actually diagnosticated. Usually there have been no very definite symptoms, and the patient has been treated for chronic gastritis. Finally these patients die, and at autopsy a wide-spreading carcinomatous ulcer is found.

Although we have pointed out the characteristics of the two main groups of cases in which cancer of the stomach is found, it is proper to dwell more at length upon certain individual symptoms. And in doing this, it is well to premise what is known to all who have anything to do with these patients, that early diagnosis of carcinoma of the stomach is so difficult as to be usually impossible; that is to say, an accurate, assured diagnosis, based on scientific reasons, and not a mere supposition nor a happy guess that the affection is malignant in nature. Even so distinguished an authority as Boas has come to the same melancholy conclusion.

The descriptions heretofore given in text books and monographs on diseases of the stomach are concerned almost wholly with the first, smaller group of cases, in which the symptoms were subacute or even acute in onset—this being the only group of cases of gastric cancer previously recognized. But as we have repeatedly pointed out, it is becoming more and more widely appreciated that this class forms only a small proportion of all patients with gastric cancer; and that the far larger group will ultimately be found to consist of those

patients who are sufferers from *chronic gastric dyspepsia*. These patients present the symptoms already noted in the section on gastric ulcer; and it is needless to reiterate those symptoms here. In the first group of patients, those who present what may be called the classical picture of cancer of the stomach, there are three symptoms which stand out with such distinctness as to be fairly characteristic; these are: pain, vomiting, and the presence of a tumor. But the pain and vomiting are not always present; and they vary greatly in their intensity, not only in different patients, but in the same patient at different times. Nor should the absence of a tumor be considered evidence that the disease is not cancer. The presence of a tumor, moreover, as already noted (p. 80), is frequently due to a purely inflammatory hyperplasia. So even in this group of cases where the clinical picture is outlined with reasonable distinctness, it is seen that diagnosis is no such easy matter.

The *pain* in cancer of the stomach differs from that of simple ulcer in several ways. It is not so sharp nor so localized a pain; it is not so invariably aroused by the ingestion of food; it is not so regularly relieved by abstinence; and it is seldom assuaged by a change of position or by rest. The pain of cancer of the stomach is more dull, aching, gnawing, and constant than is that of ulcer; it is more often of a tearing, shooting, or darting character, when perigastric adhesions are dense, or when neighboring organs (liver, diaphragm, pancreas; etc.) have been invaded by the growth.

The *vomiting* of gastric cancer depends largely upon the location of the tumor. It usually will be absent when the growth infiltrates the gastric walls widely, without obstructing the pylorus. The evidences of cardiac obstruction (see p. 176), attended by great pain, hemorrhage, and beginning cachexia, are indicative of carcinoma in this situation. When pyloric obstruction is marked, vomiting becomes frequent. At an earlier stage any indiscretion in diet may set up an acute gastritis, which, instead of subsiding as have previous similar attacks, will, in the presence of cancer, persist in a subacute or chronic form. This fact alone is suggestive of beginning cancer. As dilatation of the stomach increases, the vomiting may again become less frequent, but at the same time the amount of vomited material will become more copious, and the evidences of stagnation and fermentation will be unmistakable. Close study of the vomitus, even in the early stages, will often reveal the presence of minute quantities of clotted blood; and at all stages of the disease, tests for *occult blood* as a rule will be positive. The stools should always be examined for

occult blood. The negative value of this test is much greater than its positive value, since, as Smithies says, when it is positive, other more easily obtained and more dependable signs and symptoms of gastric carcinoma are not lacking. But if the tests for occult blood are persistently negative, cancer probably is absent. *Hematemesis* is rare; there is seldom so much blood lost as to deserve this term; or at least even if blood is lost in quantity, it usually is clotted before being vomited, and then presents the characteristic coffee-ground appearance.

Tumor is not an early sign in a pathological sense. It is, however, not unfrequently one of the first clinical evidences of the nature of the malady. Search for a tumor should be systematic and exhaustive. Palpation, with the patient erect, supine, stooping; and after the abdominal walls have become relaxed by immersion of the patient in a hot bath; percussion, with and without distention of the stomach or colon, or both, with air; and finally complete emptying of the stomach—these should all be tried, in the endeavor to ascertain the existence of a tumor. The tumor moves with respiration, and if of the pylorus or greater curvature, often possesses some lateral mobility in which case it may be mistaken for an enlarged gall-bladder. By fixing the tumor at the end inspiration, and holding it until expiration is complete, it can be felt to slide up beneath the fingers to its normal habitat in a characteristic manner. A tumor on the greater curvature becomes more evident when the stomach is distended; one on the lesser curvature disappears; one at the pylorus is pushed up beneath the liver if fixed by adhesions, while if free it descends towards the patient's right. In the presence of ascites it is of course necessary to draw off the fluid before satisfactory palpation is possible.

In addition to these three symptoms, there are three further changes constantly present in gastric cancer, which may be classed as **physical signs**: these are, *loss of weight; anemia;* and *changes in the gastric secretion.* Loss of appetite, especially for meats, arising without apparent cause, has already been mentioned as a characteristic sign; and closely following this, and caused as well by the malignant growth itself, occurs *progressive loss of weight.* To render this apparent, the patient should be regularly weighed; it is not sufficient to estimate the loss of weight from the appearance of a man's face, or his visible emaciation; the weight should be recorded periodically, not oftener than twice a week, in pounds and ounces, care being taken to avoid any errors from changes in the weight of clothing. In the case of cancer it is found practically without exception that the

loss of weight is progressive and constant, and that no form of dieting or forced feeding will check the loss permanently.

The *anemia* of gastric cancer is that encountered in carcinoma elsewhere in the body. Leriche has recently made the significant statement that if cylindrical gastrectomy were done more often for non-stenosing cancer, more cases of progressive pernicious anemia would be cured. (See Regnault: "Anémie pernicieuse et cancer latent de l'estomac." Thése de Lyon, 1905.) In gastric cancer both the red corpuscles and the hemoglobin are reduced, but rarely to the extent that one would be led to expect from the cachexia present. The leukocyte count is as a rule constantly higher than normal, the polynuclears being increased at the expense of the lymphocytes. The absence of hyper-leukocytosis during digestion is considered by some nearly pathognomonic of cancer of the stomach.

The *gastric secretion* is very constantly altered in the later stages of carcinoma of this organ. Unfortunately the characteristic changes are not early enough in their occurrence to be of material value in reaching a diagnosis for surgical purposes. These changes are: constant absence or marked diminution of the hydrochloric acid, and the presence of lactic acid and other signs of fermentation. These changes are of confirmatory value if present, but if not present, no import need be attached to their absence. In carcinoma developing on ulcer, hydrochloric acid is apt to persist; and the fermentation signs may equally well be present in stagnation from benign disease. In normal stomachs the amount of hydrochloric acid gradually increases after the ingestion of food; in cancer, no matter how small in quantity at the first test after a meal, repeated tests show that the amount grows rapidly less, instead of increasing in quantity (Gluzinsky's test). Detection, by means of Esbach's reagent, of albumen (nucleo-albumen and mucin) in the washings from a fasting stomach, after excluding the possibility of albumen being present from previously ingested food, speaks in favor of carcinoma (Salomon's test). Recently this test has been largely supplanted by the test of Wolff and Junghans. Smithies reports the Wolff-Junghans test positive in 80 per cent. of 230 cases of gastric carcinoma; but he notes that it proved positive also in a large proportion of cases of simple gastric or duodenal ulcer (66 to 78 per cent.) especially when these were accompanied by pyloric stenosis or gastric atony.

Diagnosis of carcinoma in its early stages by means of a *hemolytic blood test* has been attempted by Kelling, Crile, and others. Wideröe (1908) applied Kelling's test in 50 cases, using hen's blood exclusively:

25 of these patients had cancer, and 25 suffered from other diseases. He found that 64 per cent. of the cancer cases gave positive results; 6 of the 9 patients in whom the test was negative were already cachectic, this fact supporting Kelling's contention that the hemolysis diminishes with advancing debility. In the patients not having cancer, the hemolysis surpassed 30 per cent. in only three cases, and in 2 of these there was a disease of the blood; in the patients with cancer, however, the degree of hemolysis varied from 50 to 85 per cent. Paus, testing the blood of 90 patients, obtained a positive reaction in 65 per cent. of those in whom the course of the disease or operation confirmed the diagnosis of cancer. Crile (1908) found no hemolysis in 107 normal individuals. Among 50 diseased persons, not suffering with cancer, the test was positive in only 4 (1 with hemoglobinuria, 1 with eclampsia, 1 with hematuria, and 1 with undiagnosed gastric lesion), while among 50 carcinomatous patients, 39 presented hemolysis; and 13 out of 16 sarcomatous patients presented hemolysis. In all patients with malignant disease who did not present hemolysis, the disease was advanced. Kelling (1914) using an improved method for the test obtained a postive result in 90 per cent. (58 out of 65) of cases of carcinoma of the alimentary canal. He advocates controlling the hemolytic blood test by means of Ascole's meiostagmin reaction, since these two tests have different sources of error. If both reactions are distinctly positive, the correctness of the diagnosis is all the more certain.

Fluoroscopy has been much employed in the diagnosis of carcinoma of the stomach; but as noted in Chapter III, where the subject of the X-ray diagnosis of gastric legions is discussed, there are many sources of error, and even expert radiologists are not unanimous in their belief in the infallibility of the method.

Diagnosis.—It may now be asked: If the symptoms are so indefinite, the physical signs so misleading, and both of such late development, how is a diagnosis to be reached in time for surgery to be of any avail? This is a pertinent question; and to find a conscientious answer is the sorry duty of the surgeon. It will not do to lay the onus of this task on the physician; if cancer of the stomach is a surgical disease, the surgeon should be able to reach a reasonably correct diagnosis. Boas diagnosed sixty cases of gastric cancer within three months of the appearance of the first symptoms of the disease. He found that of these only three (5 per cent.) could be treated by resection of the growth; that thirteen were suitable for gastro-enterostomy; and that two could only be explored. Of 127 cases diagnosed within six months of the appearance of first symptoms, Boas found that only

eight (6.3 per cent.) were suitable subjects for resection. From this experience Boas came to the conclusion that the early diagnosis of gastric cancer is at present usually impossible; that the constant moan of surgery that patients are not sent early enough for operation, is not warranted by the facts; and that those patients who encounter the question of operability more than six months after the first onset of symptoms afford a greater hope of radical cure than do the earlier cases. But Hoffmann, in Mikulicz's clinique, found that of 117 cases diagnosticated within three months of onset, twenty-four (20.8 per cent.) could be treated by resection; and of 193 cases whose first symptoms dated back six months to one year, he found fifty-eight (30.3 per cent.) could be treated by resection. This discrepancy between the surgical and medical statistics is not due to the fact that Mikulicz allowed wider limits to the indications for radical operation; but because the worst cases go to the physician and the more operable ones go directly to the surgeon. Moreover, the patients seen by medical men are either hospital out-patients, or those in private practice—the number is great, but the patients do not remain long under observation; they pass on to another clinique.

It has been true in the past, but it is now much less true, at least of progressive physicians, that they did not send the patients early enough to the surgeon. The surgeon should be called in consultation as soon as an anatomical cause for the gastric disease is recognized, and in obscure cases he should be consulted even before this stage of accuracy in diagnosis has been reached. Likewise, when gastric cases come directly to the surgeon, he is only too glad to have the opinion of his medical colleagues, and to avail himself of such aids as the clinical laboratory can afford. But he has been the first to recognize that, as all signs fail in dry weather, so the possibility of reaching an accurate diagnosis must be postponed in certain instances until the patient will be beyond the help of surgery. Under such circumstances, and when there is undoubtedly some actual anatomical lesion of the stomach, even though an exact pathological diagnosis of the lesion has not been reached, but because it is evident that only some form of surgical operation will be of any avail in curing the disease—under these circumstances, we repeat, we believe exploratory operations should be undertaken. We do not advocate exploration as a therapeutic test; we do not say, do gastro-enterostomy for pain in the stomach and if the result is favorable conclude that the disease was gastric ulcer, and if the patient dies assert that death was clearly due to the cancerous cachexia; nor do we counsel exploration merely because it is easier for the surgeon

and possibly less distasteful to the patient than careful examination and repeated study of the disease by other means. Yet it is our belief that in the vast majority of patients with gastric disorders a diagnosis of sufficient accuracy can be reached before operation is undertaken thoroughly to justify the operation when done. We are convinced, moreover, that if chronic and rebellious cases of indigestion were more promptly turned over to the surgeon, and if suitable operations were done on such patients, there would be fewer cases of carcinoma of the stomach observed by physicians. It seems to us that every case diagnosticated *certainly* as carcinoma of the stomach before operation is a disgrace to the attending physician, provided he has had the patient under treatment for more than a few weeks. In that space of time it is at present usually impossible to render absolute the diagnosis of a gastric carcinoma while still in the operable stage; but it is entirely possible and we contend with all earnestness that it should be done to reach within that time the conclusion that an anatomical basis for the symptoms exists, and that this can be removed only by operative means.

In recapitulation, then, it may be said that the presence of cancer should be suspected when chronic gastric catarrh exists without any discoverable cause (such as abuse of food, of alcohol, of drugs; circulatory disturbances of the heart or liver; or diseases, such as gall stones, gastric ulcer, etc., which would cause some definite lesions in the region of the stomach), especially if this chronic gastritis be in a patient over forty years of age, and if it be attended by loss of appetite for meats (Kocher). If a tumor exists, the diagnosis is less difficult; but the tumor must be distinguished from a distended gall bladder, from a growth of the colon, of the pancreas, etc. In obscure cases distension of the stomach with air should never be neglected; this may render a hidden tumor palpable, and the characteristic pyramidal shape of a pyloric growth (apex toward the duodenum and indistinct base toward the body of the stomach) can frequently be recognized (Kocher). Occult blood in the stomach contents and feces is the most valuable of the laboratory findings. In non-malignant ulcerations of the stomach, rest in bed with milk diet will cause the disappearance of occult blood. *In cancer no treatment has any effect.*

In cases where the stomach affection resists medical treatment, exploratory operation is indicated. This is not always satisfactory, nor is it always possible, even by the senses of touch and sight, to make a positive diagnosis of carcinoma. Fortunately, the benefit is generally given to the patient and the diseased portion of the stomach removed when practicable, on the supposition that malignancy is

present. Many of the most experienced operators have been misled by the conditions present, the true condition of affairs not being realized until the excised part of the stomach was subjected to microscopical examination. If a distinct tumor is present, it can generally be recognized as carcinomatous by its irregular shape; by its "knotty" feel; by diffused induration into the surrounding structures. Before there is tumor formation of any moment, it is nearly always impossible to differentiate between the thickening and induration consequent upon inflammation and that due to malignancy. While it is true that the mortality following posterior gastro-jejunostomy in benign disease is lower than that following gastrectomy, it is always justifiable and in the opinion of some surgeons, mandatory, to subject the patient to the latter risk rather than to the surely fatal results of an undisturbed malignant condition, if such be present.[1] If there were involvement of the lymph glands with metastasis to any of the neighboring viscera the diagnosis of malignancy would be unquestioned; without these complications or extensions, in the absence of the more or less characteristic tumor formation, the diagnosis must be tentative until a final appeal can be made either to the microscope, if the affected portion of the stomach be removed, or to the extension of the disease if it be allowed to remain *in situ*. Even a microscopical diagnosis is occasionally in error. If the freezing microtome is used to facilitate diagnosis during an operation, the surgeon's duty of course is to do a radical operation, when possible, if the report from the pathologist is positive; but if negative, he must rely solely on the clinical diagnosis.

Prognosis.—Though medical means are powerless to cure cancer in any region of the body, yet no one would be so foolish as to deny that in cases of inoperable tumors much may be accomplished to prolong life and to mitigate suffering. Tyson said he was quite sure that a great deal more could be done in this way than was commonly thought possible. The wide experience of Jacobi (1906), so cautiously and conservatively expressed, in regard to the beneficial effect of methylene blue (methylthionin hydrochloride) in such patients, is a gratifying example of what may be done by medical science even for hopeless cases. But, as Bland Sutton said a number of years ago, as long as we are ignorant of the cause of cancer, so long must the only successful treatment be the extirpation of the growth.

[1] Thus Moynihan (1920) points out that the mortality from the later development of carcinoma, in Paterson's cases of gastro-jejunostomy for supposedly benign disease, is as great as would have been that from primary gastrectomy which would have cured the patient once for all.

It is sometimes questioned by physicians, whether surgery can really accomplish more in prolonging life than can medical measures. It is sometimes doubted whether surgery can ever effect a cure. To answer these questions surgeons must show the ultimate results of their operations. They must trace their patients for a period at least of three years after operation, and report their actual condition at the end of that time. But before the duration of life and the prospect of ultimate cure after surgical operation are discussed, and even before a comparison is made with the expectation of life and the certainty of ultimate death from the disease which are necessary attendants upon purely medical treatment; before these interesting questions are discussed, we repeat, it is expedient to study the immediate dangers of the operations which surgeons are urging in the treatment of this disease.

The mortality and the ultimate results of operations for benign gastric disease have already been considered in detail (pp. 101, 105). The results of such operations, which may be considered in the light of *preventive* operations for cancer, should be compared with the following figures of operations undertaken for the *cure* of this disease.

PARTIAL GASTRECTOMY FOR CARCINOMA

Operator and Reference	Cases	Deaths	Mortality per cent.
Alexinski (Centr. f. Chir., 1914, xli, 1390) (Collected statistics of Russian Surgeons.)	297	148	50.0
Altschul (Beitr. z. klin. Chir., 1913, lxxxiv, 421)	64	26	40.6
Bindseil (Inaug. Diss., Giessen, 1912)	38	8	21.0
Deaver (Records of Lankenau Hospital, Philadelphia, to 1920)	46	12	26.0
Delore and Alamartine (Lyon Chir., 1909, ii, 281)	43	14	32.5
Delore and Santy (Lyon Chir., 1914, xi, 113) 1908–11	18	3	16.6
1911–13	19	1	5.2
Feurer (Deutsch. Zeit. f. Chir., 1912, cxvi, 69)	58	11	19.0
Galpern (Centr. f. Chir., 1914, xli, 1390)	32	4	12.0
Gar (Centr. f. Chir., 1912, xxxix, 1276)	18	5	28.0
v. Haberer (Wien. med. Woch., 1912, lxii, 3089)	39	4	10.0
Küttner (Therap. d. Gegenwart, 1911, liii, 19) (Collected statistics from Breslau)	102	25	24.0
Mayo Clinic (Annals of Surg. 1919, ii, 236) (1897–1919)	736	101	13.7
Mikaye (in Jour. Am. Med. Assoc., 1914, lxxii, 2051)	116	34	29.2
Sherren (Practitioner, 1914–15, xciii, 463)	27	3	11.3
Témoin (Ann. internat. de Chir. Gastro-Intest., 1911, v, 44) Collected, 1898–1909	168	50	30.0
Before 1908	91	38	41.7
1908–1909	77	12	15.5
Weil (Berl. klin. Woch., 1913, i, 390)	135	31	23.0

This gives a total of 1580 partial gastrectomies for carcinoma, with 421 deaths, or a mortality of more than 26 per cent., as the immediate result of operations for the cure of this disease. If an early diagnosis is made, and the patient is in good general condition, the mortality is much lower. Many surgeons have a series of from 10 to 20 operations without a death. But the natural and proper desire to extend the limits of operability, while it benefits the patients who survive, serves inevitably to keep the operative mortality at the high rate just mentioned. This, it will be remembered, is to be contrasted with an immediate mortality of about 5 or 10 per cent. after operations which may be regarded as preventative of the development of carcinoma (p. 101).

It will next be proper to determine what proportion of the three-fourths of patients who survive gastrectomy may reasonably hope to be ultimately and permanently cured of their disease. Let it not be forgotten that over 70 per cent. of patients surviving the *prophylactic* operation (for benign disease of the stomach) have been proved to be ultimately cured. Robson and Moynihan in 1904 studied the statistics bearing on this point from the cliniques of Krönlein and Mikulicz; Kausch (1907) tabulated the results of Czerny and Kocher. From all these sources it is evident that patients suffering with gastric carcinoma have, under medical treatment, an expectation of life of about twelve months from the beginning of the disease. Many writers put it at less. Not only is this the duration of life, but it should not be forgotten that at the end of that time the patients under medical treatment will *all be dead*. There will not be even one among those treated medically who at the conclusion of that period will have had up to that time no recurrence of a malignant tumor successfully removed by operation, and who may, therefore, still be considered as curable—as potentially cured. If, then, surgery can show any permanent cures, and if the average duration of life under surgical treatment is longer, or at least not less than that under medical treatment, the conclusion surely is justified that surgical intervention is best for these patients. The question, in fact, is not "Is operation a sure cure for gastric cancer?" but "Does anything else offer even the shadow of a chance?" Did not Celsus write: "*Nihil interest an satis tutum sit præsidium, quod unicum est?*" Even if the surgeon is aware that one out of four patients, or four out of sixteen patients, on whom he operates for gastric cancer will surely die, he should not therefore hold his hand, and thereby condemn the whole series to certain death in about a twelve-months' time. If he could say with

certainly to himself, "I have had three recoveries from gastrectomy; this is my fourth patient, and he is therefore sure to die from the operation"—under such circumstances, of course, no surgeon would be justified in operating. But this is not the way to argue from statistics; for this surgeon with equal justice might have said to himself in the beginning, "This is my first patient for gastrectomy; even though the three following patients should recover, this first one will surely die; I will therefore refuse to operate for fear of killing him." Statistices are a valuable guide to prognosis for those who know how to use them; and no surgeon can justifiably undertake an operation which he is convinced will kill his patient; but by employing that most precious quality of mind known as judgment, and by selecting the patients who are suited for the operation in question, the skillful surgeon is enabled to save many lives otherwise doomed to destruction.

What, then, is the prognosis in regard to prolongation of life by operation? Moynihan studied the average duration of life in patients who underwent gastrectomy in the cliniques of Krönlein and Mikulicz; he found that from the beginning to the end of the disease it was as much as twenty-four to twenty-five months, or more than twice as long as the average duration of life without operation. Paterson's figures from collective statistics give the duration of life after operation (86 operations in all) as an average of nineteen months after total gastrectomy (17 patients), of twenty-two and a half months after subtotal gastrectomy (14 patients), and of just over two years after partial gastrectomy (55 patients). Kausch report an average duration of life after operation of 18.3 months in Mikulicz's patients, of 18.7 months in Kocher's patients, and of 18 months in Krönlein's patients. It should be noted that Moynihan's figures refer to duration of life after the appearance of symptoms of gastric cancer, while Paterson's and Kausch's refer only to the duration of life after operation. With this allowance, it is seen that the figures agree very closely; and as they are gathered from very different sources (Moynihan's and Kausch's from the German cliniques, and Paterson's largely from British sources), each series serves to confirm the other. Now if these patients had not been operated on, the duration of life *from the beginning of the disease* (not from the date of operation) would have been at most one year, probably less. Thus, as we have elsewhere pointed out, not only is life considerably prolonged, but at the end of this period a number of patients are still living and in good health; whereas if no operation had been done, they would all of them have been dead before this time was reached.

Moreover, not only may the mere addition to the patient's life be of utmost importance from a social, commercial, or financial point of view, but death, when it does come, will attack the sufferer in a less hideous form. The patient will not die of starvation, as he would have done had no operation been performed. And although it would be heartless in the surgeon to tell his patient, when urging operation, that even if the chance of permanent cure is slight, it is nevertheless altogether likely that he will die of cancer of the liver, with its attendant cachexia, and not from progressive starvation—although, we repeat, to draw such a picture of the future for his unfortunate patient would be diabolically cold-blooded in the surgeon, yet we doubt not that many a patient, dying of the cancerous cachexia some two years after the operation, will daily bless the art of surgery which has so prolonged his life and mitigated his suffering during the gradual approach of death.

But, though the chances of permanent cure are slight, they are not altogether imaginary. Compare the following tables: the first is reproduced from the first edition of this work, while the second represents statistics published since that date (1909).

The statistics from the Mayo Clinic, published in 1919 by C. H. Mayo, are probably the most extensive on record: not only did 37.6 per cent. of patients who survived partial gastrectomy show "three-year cures," but of 234 patients traced for more than 5 years after operation no less than 59 (25 per cent.) were still alive and well after a period of 5 years. But though these figures are very encouraging on their face, it is perhaps well to recall that the diagnosis of carcinoma in these cases presumably was based on the histological reports of pathologists whose

End Results of Partial Gastrectomy for Carcinoma (1909)

Operator	Cases without recurrence after three years		
	No. of patients	Per cent. of whole no. of operations	Per cent. of those who survived operation
Braun (cited by Creite)	2	....	6.5
British surgeons (cited by Paterson)	33	....	38.3
Czerny (cited by Kausch)	6	20.0	33.0
Kocher (Corr. Bl. f. Schw. Aerzte, 1907, xxxvii, 265)	18	19.3	26.0
Kronlein (cited by Kausch)	2	7.0	10.0
Mikulicz (cited by Kausch)	17	14.3	24.0
Robson (cited by Kausch)	..	14.0	

End Results of Partial Gastrectomy for Carcinoma (since 1909)

Operator	Cases without recurrence after three years		
	No. of patients	Per cent. of whole no. of operations	Per cent. of those who survived operation
Altschul (Beitr. z. kl. Chir., 1913, lxxxiv, 421)	64	8.0	21.0
Bindseil (Inaug. Dissert., Giessen, 1912)	5	13.0	16.6
Boeckel (Strassb. med. Zeitschr., 1910, vii, 323)	2	18.8	28.5
Deaver (Records of Lankenau Hospital)	4	12.5	17.3
Feurer (Deutsch. Zeit. f. Chir., 1912, cxvi, 69)	9	15.4	20.0
Hartmann (Presse Méd. 1919, 245)	9		
Küttner (Arch. f. klin. Chir., 1914, cv, 789)	30	18.0	26.0
Mayo, C. H. (Annals of Surg., 1919, ii, 236)	115	26.9	37.6
Sherren (Practitioner, 1914, xciii, 463)	5	18.5	20.8
Témoin (Ann. Internat. de Chir. Gastro-intest., 1911, v, 44)	33	19 6	26.9
Weil (Berl. klin. Woch., 1913, l, 390	8	10.8	13.7

findings are in dispute. It should further be remarked that not all of the specimens removed by Temoin, whose figures are quoted above, were examined, and that not all of those which were examined proved to be carcinomatous.

Possibly still further statistics might be quoted; but those already given are sufficient to show that *we may expect, at present, about* 10 *or* 15 *per cent. of patients treated by gastrectomy to be permanently cured without liability of recurrence.*

There is still another question of interest in regard to prognosis. That is the ratio of operable cases to the whole number of patients seen. W. J. Mayo writes (1913): "It may be said that with our present means of diagnosis, cancer of the pyloric end of the stomach may be recognized sufficiently early to perform the radical operation in at least half the cases." This is certainly a much higher proportion than indicated by the statistics published in the first edition of this work, which showed that up to that time surgeons had found that only in from 5 to 30 per cent. of these patients was it possible to do a radical operation.

What shall the surgeon do with those patients in whom the disease is so far advanced as to forbid a radical operation? In other words, do palliative operations prolong life and add to the patient's com-

fort? Until operation is more frequently undertaken in the very early stages of the malady, **gastro-jejunostomy** must still be the operation most often adopted. Most of the operations of gastrectomy in our own hands have been on patients in whom the existence of malignant disease was merely suspected, but not certainly known, before the abdomen was opened. A palpable tumor felt before operation will naturally suggest cancer; but the case reports already referred to (see p. 114), in which such masses have been known to disappear after gastro-enterostomy, prove that all palpable tumors are not carcinomatous in nature. As has been urged elsewhere, it is in this class of patients that exploratory laparotomy finds its most legitimate field. There was only 4 operative deaths (3 from shock and 1 from uremia) among the last 39 exploratory operations undertaken by the senior author at the Lankenau Hospital (mortality of slightly over 10 per cent.). C. H. Mayo (1919) reports from the Mayo Clinic 746 explorations, with an immediate mortality of 2.9 per cent.; while Altschul's figures give 139 explorations with 15 deaths (10.8 per cent.). The statistics from the cliniques of Krönlein and Mikulicz, studied at length by Moynihan, show that patients who had undergone an exploratory laparotomy in which no further operative treatment was possible, actually lived longer than did those whose disease was so far advanced as to make even an exploration unjustifiable, or those who entirely refused an operation of any kind. In very many operations, moreover, which are commenced as explorations merely, it is found possible either to remove the growth, or at least to perform a palliative operation which will materially prolong life and relieve suffering. This, after all, and not the production of statistics, is the end and object of surgery.

Gastro-jejunostomy for carcinoma is naturally attended by a larger mortality than are similar operations for benign disease. The tables published in the first edition of this work indicated a death rate at that time varying from 15 to over 40 per cent., the average death rate being about 30 per cent. It is probable that some reduction in mortality has been secured in recent years. The figures from the Mayo Clinic (1919) give 612 palliative operations of various kinds, with a mortality of 11.1 per cent.; Altschul (1913) reported 193 gastro-jejunostomies for gastric carcinoma,with 54 deaths (27.9 per cent.). The senior author has adopted gastro-jejunostomy for carcinoma of the stomach in 50 patients during the last 10 years (1909–1919): of these patients 7 died, an operative mortality of 14 per cent.

The following case history, of a patient who came under the care of

the junior author some years ago at the Episcopal Hospital, shows the insidious manner in which a carcinoma of the stomach may grow to inoperable size even when the patient is under medical observation; it also is an example of the striking if temporary improvement often seen when gastro-jejunostomy is adopted in cases of obstruction.

Insidious Onset of Carcinoma of Stomach; Gastro-jejunostomy for Obstruction; Patient Resumed Active Work for 6 Months

Charles G., 52 years of age, farmer by occupation.

March 21, 1915. Admitted to the Episcopal Hospital, Dr. Stevens' Service.

Lobar pneumonia (right), duration one week.

Abdominal examination: Entire epigastrium is held somewhat rigid and there is soreness on deep pressure. No abnormal masses are felt.

Has large reducible hernia on left side; wears truss.

March 30. Discharged—recovered.

December 10. Readmitted to the Medical Ward (Dr. Piersol's Service). Two days ago, while in usual health, while leading a horse, became dizzy, fell and was unable to rise, because of weakness. Never dizzy before. Taken home in cart. No pain, no paralysis, no vomiting, but he had had a bad diarrhea for a week, movements very watery with mucus and some blood. Has lost much weight recently. Chief complaint is dyspnea, abdominal soreness, and watery stools.

Lung: over right apex restricted expansion, increased voice sounds, sonorous rales and prolonged expiration. Percussion note impaired anteriorly and posteriorly.

Abdomen: slightly distended; complains of soreness over appendix and is slightly tender here. Liver not palpable below costal margin. Spleen not palpable. Left inguinal hernia as before. Blood Wassermann reaction is negative. Blood Pressure 115–85.

December 30. Discharged in good condition. Diagnosis: Chronic endocarditis.

January 17, 1916. Readmitted to Dr. Piersol's Service, complaining of vertigo, dyspnea diarrhea, and vomiting. Is somewhat emaciated. Liver extends 5 cm. below costal margin, and a mass is palpable below the costal margin in the left midclavicular line.

January 24. Test meal: Amount removed by stomach tube 100 cc.

	Microscopic:
Free HCl negative	RBC few
Lactic acid positive	WBC occasional
Total acids 43	Tumor cells negative
Free HCl and acid salts 40	Oppler Boas Bac. negative
Combined HCl 3	Sarcinæ, negative
	Parasites, negative.

January 27. Nausea improved. Tran ferred to Surgical Service (Dr. Ashhurst) for exploratory operation.

Patient has had indigestion for 2 years; is anemic; vomits occasionally (about twice weekly); typical retention vomitus, bloody; the abdomen is thin; there is a barely palpable mass in the epigastrium. Diagnosis (Dr. Ashhurst): pyloric stenosis.

January 28. *Operation* (Dr. Ashhurst), Ether. A large mass, evidently malignant, was found in body of the stomach, not involving pylorus or cardia, but extending from greater to lesser curvature on both anterior and posterior walls, and almost completely obstructing its lumen. There were numerous enlarged rather soft lymphnodes in the gastrocolic omentum; fewer and smaller lymphnodes in the gastrohepatic omentum. The

stomach was not fixed, but the tumor was considered inoperable on account of its extension so far toward the fundus of the stomach, and on account of the patient's weakness. No metastases were found in the liver. The gall-bladder was normal. With great difficulty enough of the posterior wall toward the fundus of the stomach was exposed through the transverse meso-colon to render a gastro-jejunostomy possible; the anatomotic opening was less than 5 cm. in length. After operation enteroclysis was given.

January 29. Water by mouth. Vomited a little blood while coming out of ether. No unfavorable symptom since.

February 1. Liquid diet.

February 4. Semi-soft diet.

February 12. Sitting up. Feels very well. Soft and extra diet.

February 16. Walking around ward.

February 19. Went home in good condition.

September 15. Readmitted to Dr. Ashhurst's Service. Has been in perfect health since operation. For the last 5 months has worked as farmer (plowing, hoeing, harrowing, digging, etc., as usual), with no disability and never a symptom from his stomach.

Returns today for abdominal pain and constipation of 3 days duration. Has had a bowel movement every day. Abdomen: A firm epigastric mass moving in respiration, extending almost to umbilicus. Irreducible (incarcerated) left inguinal hernia. Diagnosis. epiplocele.

Operation (Eucain 1 per cent.) by Dr. Ashhurst. Nodules of metastatic carcinoma found in an otherwise empty hernial sac. Omentum perhaps spontaneously reduced. Much yellow (not bloody) ascitic fluid drained from internal ring before closing wound. Many hard nodules felt in omentum by finger passed through internal ring. Sac not removed.

September 22. Uneventful recovery. No vomiting. Appetite excellent.

September 23. X-Rays show tumor obstructing most of stomach, with only pencil sized channel through to pylorus. Stomach empties in normal time.

September 29. Went home in good condition. Weight 138 pounds with clothes.

November 4. Readmitted to Medical Ward, Dr. Robertson's Service. Chief complaint: weakness and nervousness. Ankles began to swell last week, and began to get puffy about the eyes. Some dypsnea, frequency of urination, and gradually increasing distention of abdomen.

November 8. Parancentesis abdominis: 2140 cc. of clear amber fluid withdrawn. Paracentesis of chest: 90 cc. of similar fluid.

December 8. Paracentesis abdominis repeated: 1097 cc. of similar fluid withdrawn. Dyspnea relieved.

December 18. Paracentesis of right chest: 3600 cc. of similar fluid withdrawn.

December 20. Patient allowed to go home unimproved. Death occurred in the following February.

The average duration of life after gastro-jejunostomy for malignant pyloric obstruction is almost certainly longer than when no operation has been done; but so far as we have been able to ascertain, the details thus far have been published in too few cases for very positive conclusions to be drawn. Moynihan traced 26 out of 30 patients who recovered after gastro-jejunostomy for cancer; six patients were still alive, one after thirteen months, and five less than twelve months since the operation. Of the 20 patients who had died, the shortest duration of life after operation was fourteen weeks; while two patients had lived more than two years. In Krönlein's patients who recovered

from gastro-jejunostomy and were traced (54 in number) the average duration of life after operation was 193 days (over six months), and in Mikulicz's patients it was 6.4 months—or in each series about 3 months longer than if no operation had been employed. Moynihan, however, notes that if in this reckoning the immediately fatal cases are included, the average duration of life is slightly less than if no operation had been employed. However, the statistics of Mikulicz were compiled in 1901, and those of Krönlein in 1902; and it is but reasonable to suppose that since that time some improvement even in the results of gastro-jejunostomy for carcinoma has occurred.

It appears from these statistics that in the hands of experienced abdominal surgeons the immediate mortality from gastro-jejunostomy in patients with cancer of the stomach is as high as, and in some instances even higher than that of partial gastrectomy for the same disease. But even if these statistics represent correctly the practice of the present, which is a little doubtful, it is not probable that this difference in the mortality of gastrectomy and gastro-jejunostomy for cancer is due to any inherent qualities of the respective operations; it seems rather attributable to the fact that gastro-jejunostomy has been and is still employed in patients already nearly dead from starvation and cachexia, with the forlorn hope of relieving their discomfort during their remaining days on earth. On the other hand, the majority of surgeons have been fearful of employing so extensive an operation as even partial gastrectomy in any but carefully selected patients. We believe that, other things being equal, gastrectomy is the more serious operation of the two; and were it to be used as indiscriminately as gastro-jejunostomy has been, the relative mortality rates would appear in true proportion. Oversight of this fact seems to give some basis for the enthusiasm with which Robson and Moynihan suggest the employment of gastrectomy as a palliative operation even in cases where it is manifestly impossible to remove the entire disease. They speak as follows of the results of gastro-jejunostomy in cases of pyloric obstruction from cancer: "There can be no doubt that in such cases gastro-enterostomy is productive of the most remarkable benefit to the health and well-being of the patient. The weight increases, the appetite and the power of gratifying it return, and vomiting, often the most distressing and unceasing symptom, stops at once. But there can also be no doubt that in some instances, when the growth does not actually obstruct by its bulk the onward passage of food, a decided benefit results from the operation." But they say later, "The question may arise as to whether gastrectomy should not be per-

formed deliberately as a palliative operation in cases where an early secondary deposit can be seen in the liver, or inaccessible or irremovable glands be found in the pancreas, or along the aorta and vena cava. If we take into account the following advantages of gastrectomy as compared with gastro-enterostomy—that in the most competent hands its mortality is not greater, but is even less, than the mortality of gastro-enterostomy; that a prolongation of life for ten months longer than the period given by gastro-enterostomy is the rule; that the comfort, the general health, appetite, and well-being of the patient are all emphatically better; and, finally, that the patient has always a chance, even though it is of the slenderest, of a complete recovery from his disease—if we take all these into our consideration, there can be no doubt that the operation of choice will always be gastrectomy." And W. J. Mayo (1914) writes that "patients subjected to the removal of the visible growth in the stomach, even if all the glands cannot be removed will get a year or more on the average of a very comfortable existence cheered by the knowledge that there is a possibility of cure, since, in some cases, irremovable glandular hyperplasia is the result of infection rather than metastasis." Nor should the remarkable results achieved by Témoin be overlooked: this surgeon makes a practice of removing the stomach and leaving behind all enlarged glands—wiping the stomach away from its omental attachments by dry gauze dissection. In spite of this partiality of these experienced surgeons, it seems to us that gastrectomy is best reserved for those cases in which it seems likely that it will be a curative operation. Surely if employed when secondary deposits exist in the liver, or where there are inaccessible or irremovable, but nevertheless surely carcinomatous lymph nodes—surely in such cases there can be not even the slenderest chance of a complete recovery from the disease after gastrectomy.

Finally, it is of interest in this connection to recall the researches of Katzenstein (1906), as the result of which he suggested that the arrest of carcinomatous growths sometimes observed after gastro-jejunostomy might be due to the local action of the trypsin of the pancreatic juice, freely admitted to the stomach after the usual lateral anastomosis employed in this operation.

Prognosis after Gastrostomy and Jejunostomy.—Still other palliative operations may be employed: gastrostomy in patients with carcinoma of the cardiac orifice, and jejunostomy where the pylorus is obstructed and the stomach is too extensively diseased for the performance of gastro-jejunostomy.

The following tables show the mortality in several series of cases of *jejunostomy:*

JEJUNOSTOMY FOR BENIGN DISEASES OF THE STOMACH

Operator	No. of operations	Deaths	Mortality per cent.
Bérard (1912)	3	0	0.0
v. Eiselsberg (1914)	24	12	50.0
Haudek (1914)	6	3	50.0
Remijnsee (1912)	10	1	10.0
Simon (1913)	13	6	46.0

JEJUNOSTOMY FOR MALIGNANT DISEASES OF THE STOMACH

Operator	No. of operations	Deaths	Mortality per cent.
Altschul (1913)	5	4	80.0
Bérard (1912)	7	6	85.7
Hoffman (1911)	10	8	80.0
Küttner (1911)	21	10	50.0
Remijnsee (1912)	23	8	34.0
Spencer (1910)	5	4	80.0

It should be noted, however, that the figures cited above do not refer in all instances to the immediate (operative) mortality, but include also deaths following within a few weeks or months of the operation. But the operation of jejunostomy *per se* and quite apart from the patient's general condition, has a distinct mortality of its own, evidenced by the fact that death in not a few instances has been due to intestinal obstruction from occlusion of the afferent jejunal loop by kinking, rotation, strangulation over it of other coils of intestine, etc.

Duodenostomy (above the bile papilla) has been employed successfully by Hartmann, and he advocates it as in every way superior to jejunostomy. We have had no experience with it.

It is our opinion that such palliative operations as these are very rarely indicated. It is very unusual, as Dawson reminds us, for thirst to be an annoying symptom of carcinoma of the esophagus or of the cardiac orifice of the stomach; and we consider his strong condemnation of such meddlesome surgery fully justified by the trend of thought today. So long as patients with inoperable internal carcinoma are not starving to death, it is the part of wisdom to refrain from palliative and useless operations.

Treatment.—Having pointed out in the section on Prognosis, the expectation of life and the hope of radical cure which operative treatment offers to patients with carcinoma of the stomach, it next becomes necessary to decide upon the special type of operation to be adopted in specific cases of the disease.

The terminology of gastric surgery is not entirely uniform throughout the surgical world. The terms as used in this volume are defined in the chapter on the Technique of Operations on the Stomach (Chapter XIV), and to that the reader is referred for detailed descriptions.

Total gastrectomy will very rarely be advisable. Such extensive invasion of the gastric wall as to make this operation requisite will usually be found to be accompanied by so many perigastric adhesions or by such obvious metastases, as to render useless any but a palliative operation. Though successful in a sufficiently large number of cases to remove the procedure from the realm of mere surgical experiment, it is not an operation which any surgeon should feel himself competent to undertake, save one who has been thoroughly trained in gastro-intestinal surgery. Trinkler (1911) collected 26 cases with 9 deaths (34.6 per cent.).

Subtotal gastrectomy (Fig. 112) is more difficult than partial gastrectomy only where adhesions abound. If there are extensive adhesions to the pancreas, any form of gastrectomy must usually be inadvisable. Although in a few cases portions of the pancreas have been excised in one mass with the stomach, yet the danger from infection, and from the digestive action of the pancreatic juice is so great, that the surgeon is rarely justified in exposing his patient to the greater risk, especially as freedom from recurrence in these cases is not to be anticipated. The raw surface of the pancreas usually must be covered in with gauze packs, and the patient's convalescence is thus much more delayed than when the abdominal incision can be completely closed. Yet Childe successfully excised a layer of pancreatic tissue in one piece with the stomach and the transverse colon, and Sauvé has published (1908) a paper advocating partial pancreatectomy when necessary.

Excision of the transverse colon *en masse* with the cancerous stomach appears to have been employed at least in 39 cases, with 26 recoveries and 13 deaths; the mortality (33.9 per cent.) is thus considerably less than might have been expected from so extensive an operation. Leriche (1906) collected 31 cases, and at least 8 additional cases have been recorded during the last decade. It is an operation which is logically correct, when the transverse colon is itself invaded, but not to such an extent as to prevent entire removal of the malignant

growth; and even in cases where the blood supply of the colon is jeopardized by the radical removal of the gastric disease (as in Childe's patient, referred to above, and in Krause's patient), even though the colon be not itself invaded by carcinoma, it is better successfully to remove an organ whose blood supply is destroyed, than to expose the patient to almost certain death from gangrene of the colon. In one patient under the care of the junior author at the Episcopal Hospital, it was found after partial gastrectomy for a large pre-pyloric tumor (afterwards proved to be inflammatory) that the blood supply of the tranverse colon had been injured in detaching the adherent posterior gastric wall; the transverse colon was therefore resected, an end-to-end anastomosis being done. Recovery was uneventful (Figs. 30, 31). In a few instances (Massmann, Ross, and others), the middle colic artery has been unwittingly ligated in doing a gastrectomy, and the patients have died from peritonitis due to the ensuing gangrene of the transverse colon.

Partial gastrectomy is the operation of choice for the radical cure of gastric carcinoma. As already remarked the differences between it and subtotal gastrectomy are slight, in regard to difficulty of performance, immediate mortality, or operative technique. Subtotal gastrectomy is necessitated by a more extensive invasion of the neoplasm toward the fundus and along the greater curvature. The question of most interest in this connection, in regard to partial gastrectomy, is the method to be adopted for restoring the continuity of the gastro-intestinal canal. These methods are Billroth's first method; Billroth's second method; Kocher's method; and posterior trans-mesocolic gastro-jejunostomy—which latter procedure includes several subvarieties, as the long loop method (with or without entero-anastomosis), the no loop method, the Y-method, Polya's method, etc. Full descriptions of these various methods will be found in Chapter XIV.

Billroth's first method is nearly universally condemned. According to Paterson leakage at the "fatal angle" occurred in 23 per cent. of the cases he collected. Kocher quotes Guinard's collective statistics, of 148 gastrectomies by Billroth I, with a mortality of 35.3 per cent. After mere pylorectomy, however, the mortality is lower than this (p. 278).

Billroth's second method (anterior gastro-jejunostomy), though inferior in our judgment to an operation completed by a posterior trans-mesocolic gastro-jejunostomy, is more widely applicable than the latter, in cases of subtotal gastrectomy, when the cardiac pouch is small. Unless it is contra-indicated, our preference is for the

restoration of the gastro-intestinal canal by posterior trans-mesocolic gastro-jejunostomy, by the "no-loop" method or by Polya's method. Difficulty of performance, as after subtotal gastrectomy, is the chief, indeed almost the only valid, contra-indication.

Kocher's method was of course strenuously supported by its author. He quoted Guinard's collective statistics of 64 operations by this method, with a mortality of 15.6 per cent. In the hands of Kocher and his associates (1907) this operation had been employed 92 times, with 14 deaths, a general mortality of 15.2 per cent. (12 deaths among the first 71 operations, or a mortality of 16.9 per cent.; and 2 deaths among the last 21 operations, a mortality of only 9.5 per cent.). Kocher further calls attention to the fact that all but three of the patients permanently cured were operated on by this method; of these three, one patient was operated on by Billroth's first method; and in two patients the operation was circular (cylindrical) gastrectomy.

Polya's method, in which a lateral anastomosis (transmesocolic) is made between the upper jejunum and the open stump of the stomach, is especially valuable after subtotal gastrectomy, where the pouch of stomach which remains is very small or difficult of access.

The operations of **cylindrical gastrectomy** and of **gastric resection** are very rarely indicated in cases of malignant disease. In carcinoma involving only the median portion of the stomach, the former may sometimes be available, but the rule enunciated by Leriche (1907), himself one of the chief advocates of cylindrical gastrectomy, should be strictly enforced—namely, that cylindrical gastrectomy is *absolutely contraindicated* if there are enlarged glands in the gastro-hepatic omentum; since under these conditions it is imperative for the surgeon to remove the entire lesser curvature. Reidel (1909) reports a series of 23 cylindrical gastrectomies for *benign* disease, with a mortality of 26 per cent. In cases of carcinoma the death rate probably is higher.

Enderlen's experience, reported by Faulhaber and Rewitz (1914), probably is exceptional: in his service there have been done 26 cylindrical gastrectomies, with only one death, due to leakage at the suture line.

Gastric resection is suitable only for tumors confined to the anterior or the posterior wall of the stomach, and involving neither curvature; or for those confined to the greater curvature alone, near the fundus. How extremely rare such growths are, is recognized by all; and even should it seem possible to adopt this operation, the execution of a

typical partial or subtotal gastrectomy would not only prove easier in most cases, but would offer a greater chance of ultimate cure.

The following figures from the Mayo Clinic have been published by C. H. Mayo (1919).

Operation	No.	Mortality per cent.
Billroth I	19	5.0
Billroth II	359	12.5
Kocher	7	14.2
Cylindrical	28	14.2
Posterior Polya	115	14.7
Anterior Polya	120	13.3
Resections	12	25.0

Gastrostomy is applicable only to cancer of the cardiac orifice or of the esophagus. We think it should seldom or never be employed in the case of patients who can still swallow liquids.

Jejunostomy or **Duodenostomy** may occasionally be adopted in cases of diffuse infiltration of the gastric walls not admitting of gastro-jejunostomy. In employing either gastrostomy or jejunostomy the precarious state of the patient must be kept in mind; it is best for the surgeon to know before beginning the operation just what he intends to do, and then to do it without any unnecessary intra-abdominal explorations. By heeding this advice, and by adopting these operations as soon as a diagnosis is made, instead of waiting until the patient has one foot already in the grave, the surgeon may expect his immediate mortality to be almost *nil*, and his patients really to derive some benefit from the operation.

CHAPTER XII

CARCINOMA OF THE DUODENUM; SARCOMA AND ENDOTHELIOMA OF THE STOMACH AND DUODENUM

Carcinoma of the Duodenum is rare. Deaver and Ravdin (1920) collected records of 151,201 autopsies, among which carcinoma of the duodenum was found 50 times, or in 0.033 per cent. of cases. G. Jefferson (1916) noted that of 4177 cases of intestinal carcinoma, only 130 or 3.1 per cent., were situated in the small bowel; or in other words, that 96.9 per cent. of the cases occurred in the colon or rectum. But among 132 cases of carcinoma of the small intestine in which the location of the tumor was definitely stated, 63 or nearly 48 per cent. were in the duodenum, and 69 in the jejuno-ileum (Deaver and Ravdin); considering the relative length of the duodenum to the remainder of the small bowel, it is seen that there is a very striking predilection for the former to be affected, especially when it is borne in mind that these figures *do not include carcinoma of the papilla of Vater.*

Duodenal carcinoma usually is described according to the relation it bears to the orifice of the bile duct in the duodenum, as supra-ampullary (para-pyloric), juxta-ampullary (peri-ampullary), and infra-ampullary (juxta-jejunal). Of these forms the juxta-ampullary is the most frequent; it is not considered in this place, but in connection with obstruction of the bile-ducts (p. 506). Among 158 cases of duodenal carcinoma collected by Deaver and Ravdin (1920), 35 (22.15 per cent.) involved the first part; 104 (65.82 per cent.) were in the second or descending portion; and 19 (12.02 per cent.) in the third (transverse) portion. Forgue and Chauvin (1914) succeeded in collecting 45 cases of (non-ampullary) carcinoma of the duodenum, in 29 of which the site of the growth was given: 17 were supra-ampullary, 11 infra-ampullary, and in 1 case the involvement was diffuse.

Little is known of the etiology of duodenal carcinoma. Jefferson (1916) found records only of 30 cases of carcinoma of the duodenum which certainly followed ulcer, and even in some of these the existence of a preceding ulcer is assumed rather than proved. Houdard (1913), according to Jefferson, thinks the rarity of carcinoma following ulcer of the duodenum is due to the rare occurrence of callous ulcers in this situation; this in turn being due to the absence of the thick submucosa which in the stomach forms such a favorable site for the development of inflammatory thickening around the base of the ulcer (Plate I).

Several cases are quoted by Jefferson (Sherren, Mayo, Bland Sutton) where an ulcer of the duodenum has extended into the stomach and has become malignant only in its gastric portion.

Most duodenal carcinomata are of the cylindrical celled variety (adenocarcinoma). The growth, especially when adenocarcinoma, is apt to constrict the intestine, and symptoms of obstruction are therefore prominent. When viewed from outside the gut, the appearance is as if a "string had been tied tightly around the bowel" (Fenwick). If above the bile papilla the symptoms of pyloric obstruction are so closely simulated as to make a differential diagnosis impossible. When upon, or in the immediate neighborhood of the bile-papilla, chronic jaundice is present, and differentiation from other causes of biliary obstruction becomes important (p. 506). An accompanying dilatation of the stomach, usually absent in affections confined to the biliary and pancreatic tracts, may be an important differential sign. Below the orifice of the common bile-duct, obstruction of the duodenum is less difficult to recognize; the nearly constant presence of bile in the vomitus is a most important symptom. If this were due to a gastro-biliary fistula, instead of to regurgitation of bile from the duodenum, the vomitus would not be found to contain the pancreatic ferments as well as bile. Finally, in no case should fluoroscopic examination be omitted.

Treatment should be by operative means, which often are requisite for a correct diagnosis. Unfortunately in most cases only a palliative operation (gastro-jejunostomy) can be performed. Syme (1904) resected three inches and a half (9 cm.) of the third portion of the duodenum for an annular carcinoma, restoring the intestinal canal by end-to-end anastomosis of the duodenum by suture; his patient recovered and was in good health three months later. Forgue and Chauvin (1914) mention 3 other radical operations in addition to that recorded by Syme, with 2 successes.

In a patient at the Lankenau Hospital, the senior author operated for symptoms of 4 mos. duration, especially vomiting and epigastric pain; he excised from the juxta-pyloric portion of the duodenum a small nut-sized tumor which was found on microscopical examination to be carcinomatous; the growth was of the adenomatous type, seemingly derived from the pancreas, of which tissue there were some areas in the section. The patient was in good health one year after the operation, but has not been traced subsequently.

In a second patient with carcinoma of the duodenum, under his care in the University Hospital (1919), whose history he has reported (1920) in association with Dr. I. S. Ravdin, operation was undertaken

upon a diagnosis of carcinoma of the stomach: a fixed mass was found arising from the posterior inner wall of the duodenum, below the papilla of Vater, near the termination of the second portion. After mobilization of the duodenum it was found that the ulcerating mass had invaded the pancreas and become inoperable. A posterior gastro-jejunostomy was done but the patient died suddenly of acute dilatation of the heart 48 hours after operation.

Sarcoma of the Duodenum.—Fenwick (1902) referred to 21 cases. Obstruction is rare, the tumor being rather soft and vascular than constricting. Fatal hemorrhage has been noted (Rolleston). Angier and Feivez (1912) report a case in a child aged four years and a half; death occurred soon after operation.

Sarcoma of the stomach has been considered a rare disease; but Fenwick (1902) observed two examples of round celled sarcoma among "twenty-three consecutive autopsies upon persons who had died from primary neoplasms of the stomach," and Venturelli (1915) has collected 172 cases of gastric sarcoma. Some of the cases recorded as such probably are not true sarcomata. Hosch (1907) found among 13,387 autopsies, recorded in the University of Basel, that there were 168 instances of sarcoma; and that 6 of these (3.5 per cent. of the sarcomata) were primary in the stomach. The metastatic is much less rare than the primary form.

Yates (1906) found from a study of the literature that from 28 to 45 per cent. of the reported tumors were of the round cell variety, from 32 to 36 per cent. of the spindle cell form, while lymphosarcoma constituted from 15 to 35 per cent. of cases. Mixed tumors are frequent, among those encountered being the myxomatous and the angeiomatous; fibrosarcomata and myosarcomata also are not unusual. Cysts may form from hemorrhages or necrotic processes in the central parts of the tumor. Indeed it is not infrequently impossible even for a skilled pathologist to determine whether such a tumor is a myoma, a myxoma, an angeioma, or a true sarcoma, or even an endothelioma; and when the existence of a mixed form is indubitable, it may be impossible to say whether the angeioma, the myoma, etc., was the primary growth and was originally benign, or whether the tumor was malignant (sarcomatous) *ab initio*. This uncertainty was noted when describing benign tumors of the stomach, and is merely recalled here in passing, since any discussion of moot points in pathology would be out of place in a work of this kind.

The sarcomatous growth usually commences in the submucous tissues, and the mucosa may remain intact for a considerable time.

The tumor frequently attains a considerable size, and may project into the cavity of the stomach or may protrude beneath its serous coat, sometimes invading the gastro-hepatic or the gastro-colic omentum.

Gastric sarcoma has been found, according to Yates, at the cardiac orifice in 6 per cent. of cases, at the fundus in 58 per cent., at the pylorus in only 36 per cent. (compared to 60 per cent. of carcinomata found at the pylorus); and no more than 9 per cent. of those at the pylorus produced obstruction. About one-third of the tumors were more or less diffuse. Yates found that metastasis was noted in 70 per cent. of the round cell sarcomata and lympho-sarcomata, and in less than 50 per cent. of the spindle cell variety. The kidneys (in 28 per cent.); the liver, ovaries, pancreas, adrenals and omentum (each in 14 per cent.); and the lungs, diaphragm, pleuræ, esophagus,intestine, and mesentery (in about 7 per cent.), are the organs most often involved (Fenwick). The skin presented metastatic nodules in about 12 per cent. of the reported cases. Hosch (1907) calls particular attention to the fact that whereas sarcomata in general (throughout the body) give metastases to the liver in 40 per cent. of cases, those which were primary in the stomach produced secondary hepatic growths in only one-tenth of the recorded cases.

Symptoms.—The symptoms due to the presence of a malignant growth, namely, anemia, emaciation, etc., are similar to those encountered in patients with gastric carcinoma; but pyloric obstruction, which is usual in the latter disease, is seldom seen in cases of sarcoma of the stomach; and a history of long standing dyspepsia is also rare. Tests of the gastric secretion give results similar to those obtained in carcinoma. Fenwick lays stress upon the great enlargement (non-malignant) of the spleen, in 15 per cent. of patients with sarcoma of the stomach; this enlargement, when present, is an important differential sign. Perforation is said to be more frequent (11 per cent.) than in carcinoma of the stomach. Hemorrhage is characteristic of the angeiomatous and myomatous forms. Carcinoma and sarcoma have been found associated twice in the stomach.

Diagnosis from carcinoma is rarely possible before operation, and often a distinction can be reached only by microscopical examination. The age of the patient is an unreliable guide: among 70 cases where the age was recorded, Hosch found 27 patients under 40 years of age, 26 over 50 years of age, and 17 (the greatest number in any decade) between 40 and 50 years. The very marked prostration of strength, the excessive anemia, and the early occurence of slight but persistent pyrexia, all speak in favor of sarcoma. When the

Endothelioma Springing from Pylorus. Specimen Removed by Partial Gastrectomy. Above can be seen the Pyloric Portion of the Stomach with its Sectioned Surfaces and Pouting Mucosa; Between them the Remains of the Gastrohepatic Omentum. Below is the Tumor, Springing in Three Great Masses from the Greater Curvature Beneath the Pylorus. Female 46 Years. Recovered, and in Good Health One Year after Operation. Path. No. 12,776. *Lankenau Hospital.*

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abdomen is opened, one would be inclined to diagnosticate sarcoma from the existence of a well defined tumor, especially if of rather large size, not obstructing the pylorus, softer and more vascular than a a carcinoma, situated on one of the gastric walls or at the fundus, rather than along the lesser curvature; and from the absence of perigastric adhesions.

Prognosis.—Without operation the average duration of life is "fifteen to eighteen months for the round cell, and twenty-four to thirty-two months for the spindle cell forms, both distinctly longer than carcinoma." (Yates.)

Treatment.—If a satisfactory diagnosis cannot be reached within a few weeks, exploratory operation should be urged. Haggard (1920) has collected 107 cases in which operation was done—among 58 partial gastrectomies there were 15 deaths (25.8 per cent.), and among 10 palliative or merely exploratory operations there were 8 deaths (80 per cent.); the results of the other operations are not recorded. The heavy mortality in cases in which exploration only was done, no doubt is to be attributed to the precarious condition of the patients. More ancient series of operations, however, gave a higher mortality for radical operation: thus Zesas (1913) collected 40 radical operations, with 15 deaths, a mortality of 37.5 per cent.

The prospect for permanent cure is not very favorable: in 12 cases referred to by Frazier (1914) in which the subsequent history is known, 1 survived 14 years, 2 for 2 years, 1 for 1 year, 1 had recurrence 3 years after operation, and 8 were reported well from 2 to 11 months after operation. Hesse's patient (1912), not included in Frazier's statistics, was reported living and well 7½ years after operation.

Endothelioma of the Stomach.—Jörgensen (1911) cites 5 cases of this nature, and a few have been reported since. Both clinically and microscopically it resembles sarcoma. We have references to 6 operations for endothelioma of the stomach. Morris (1910) did gastro-jejunostomy for an inoperable tumor, the diagnosis being made from a section removed at operation; Stauder (1908), Maylard and Anderson (1910), Jörgensen (1911), Schiassi (1912), and Sherrill (1915) did radical operations; 4 of the patients are said to have survived the resection; Jörgensen's patient was still free from recurrence 10 months later; Stauder's patient developed symptoms of recurrence 4 months after operation; and the late history in the other patients is not recorded.

In the case of the patient whose stomach forms the subject of Plate IV, partial gastrectomy was successfully done by the senior author, but the patient cannot be traced.

CHAPTER XIII

INJURIES OF THE DIAPHRAGM, STOMACH AND DUODENUM

Of the various injuries to be considered in this chapter, the majority are rare occurrences in civil hospitals. The nationality of patients has also a considerable influence in determining the relative frequency with which stab-wounds and gunshot wounds are encountered, while subcutaneous ruptures are almost exclusively confined to city hospitals with large accident services. Stab-wounds are therefore more frequent in southern Europe, and among Italian laborers in this country, than in other portions of our population; while gunshot wounds are rare in northern cities, except from negro brawls, compared to their incidence in the southern states, where the experience of one surgeon may embrace a hundred or more operations for such injuries.

I. Injuries of the Diaphragm.—These are of interest to the abdominal surgeon because of the frequency of abdominal complications.

(A) Stab-wounds.—These usually result from penetration of one of the lower (sixth to tenth) intercostal spaces on the patient's left. The lung is rarely injured, but the dagger, if it penetrates the diaphragm, not unfrequently punctures the stomach, the colon, or the omentum. Among 73 operations for wounds of the diaphragm, analyzed by Suter, there was injury of the abdominal contents in 24 cases, or 33.33 per cent.

The *symptoms* are chiefly those of shock and hemorrhage. The omentum frequently, and the stomach or colon occasionally, protrudes in the thoracic wound; this is of course pathognomonic of penetration of the abdominal cavity. The physical signs closely resemble those of diaphragmatic hernia (see p. 239). Operation should be undertaken before sufficient time has elapsed for evidences of peritonitis to appear.

The *prognosis* without operation is very bad. Sorrentino (1895) referred to 33 cases in which no operation was done. Of these patients, 29 died, a mortality of 87.8 per cent. Among the 29 deaths, 15 occured soon after the injury, giving an immediate mortality without operation of 50 per cent.; the other 14 patients lived for months or years, and then died from conditions which prompt operation can pre-

vent. The causes of death in the patients who died soon after injury were as follows:

Incarceration of abdominal organs in wound of diaphragm	7 patients.
Extravasation of gastric contents into pleural cavity	5 patients.
Hemorrhage	1 patient.
Empyema	2 patients.

Of those patients who survived their injuries for the time being, every one reported died later from incarceration of the diaphragmatic hernia. Although these figures are not very recent, they may be accepted as a fair indication of what the outcome of these cases must be if no operation be employed, as the non-operative treatment of such cases has changed little, if at all, since these statistics were collected.

On the other hand, the results of operation are very encouraging. Lenormant in 1903 collected 31 operations for wounds of the diaphragm, with only 8 deaths, a mortality of 25.8 per cent. Suter in 1905 analyzed 79 such operations; 70 patients recovered, a death rate of 11.4 per cent. Of the 9 fatal cases, only 2 were unaccompanied by injury of the abdominal viscera. Salomoni (1910) studied 229 operations for stab wounds of the diaphragm: only 46 patients died, a mortality of 20 per cent.

Treatment.—It being decided that operation is indicated, it next becomes necessary to determine what the operation shall be; whether thoracotomy, laparotomy, thoraco-laparotomy, or a combined operation (an operation which opens both pleural and peritoneal cavities by means of a single incision).

Operations for Stab-wounds of the Diaphragm (Salomoni)

Operation	No. of cases	Recovered	Died	Mortality per cent.
Thoracotomy	163	148	15	8.6
Laparotomy	39	21	18	46.0
Thoraco-laparotomy and "combined operation"	27	14	13	50.0
Total	229	183	46	20.0

The much higher mortality in cases treated by laparotomy is not due, as might be supposed at first sight, to graver injuries than in the cases of patients treated by thoracotomy. Suter (1905) gave the following figures for 24 operations in which injuries of the abdominal viscera were present:

Operations for Stab-wounds of the Diaphragm Complicated by Injuries of Abdominal Viscera

Operation	No. of cases	Recovered	Died	Mortality per cent.
Laparotomy	9	5	4	
Thoraco-laparotomy	2	1	1	50.00
"Combined operation"	1	0	1	
Thoracotomy	12	11	1	8.30

J. D. S. Davis (1914) reports a remarkable series of four operations for recent stab wounds of the diaphragm, all of the patients recovering: in three the diaphragm was repaired by thoracotomy and the abdomen was immediately opened for exploration; in one patient no abdominal lesion was found; in the second a wound of the stomach, and in the third one of the colon was repaired. The details of operation in the fourth case are not recorded.

The technique of the operation is essentially the same as that for diaphragmatic hernia, which has already been described (p. 242). By laparotomy it is usually very difficult, if not impossible (1) to reduce the herniated organs, owing to the negative pressure within the thorax; (2) to repair the wound in the diaphragm; (3) to suture wounds of the cardia or the fundus of the stomach. If the herniated viscera can be reduced from within the abdomen, pneumothorax is produced by this procedure quite as surely as by the operation of thoracotomy. By the latter operation the injured viscera can be quite satisfactorily repaired, as well as the wound in the diaphragm; or if there seem good reason to believe that further abdominal injuries exist, inaccessible to treatment from above, the abdomen may be opened subsequently as was done by Davis in the cases mentioned above. Pleural infection occurred, according to Suter, in less than 10 per cent. of the whole number of operations.

(B) **Gunshot wounds** of the diaphragm have no interest apart from the accompanying visceral injuries. Yet Robert (1903) reported the case of a patient who died from strangulated diaphragmatic hernia one year after a gunshot perforation of the diaphragm, from which he had recovered without operation; and a similar case came under the notice of the junior author at the Episcopal Hospital some years ago: Operation was done by another surgeon for symptoms of acute intestinal obstruction, the operation being abandoned when no cause of obstruction could be found; and the significance of the history

of a gunshot wound in the thorax many years previously was not appreciated until at autopsy a strangulated diaphragmatic hernia was found.

(C) **Rupture of the Diaphragm.**—*Subcutaneous* as distinguished from *percutaneous* injuries of the diaphragm are extremely rare. Iselin (1907) advises, and we believe justly, that primary laparotomy be the operation of choice in these cases, since extensive lesions of the abdominal viscera are more frequent than in the case of stab-wounds, and hemorrhage from ruptured blood-vessels may be inaccessible by the thoracic route. If it be difficult to reduce the herniated organs, the surgeon may create a pneumothorax by a small intercostal incision, whereupon reduction will be easy. After the more serious lesions have been repaired (hemostosis, suture of gastro-intestinal perforations, etc.), and if the patient's condition permit, the surgeon may open the thorax and suture the rent in the diaphragm from above. If collapse of the patient prevent this step of the operation, the rupture should be tamponned from the abdominal wound, in the hope that protective adhesions may form. Omentum, and even liver or stomach might be sutured in place to stop the gap.

It has been possible to find references only to five operations for subcutaneous rupture of the diaphragm. Two patients recovered.

1. Guibal. Laparotomy by Villemin: herniated organs reduced; profuse hemorrhage; wound hastily tamponned. Patiend died in a few minutes after return to bed. Autopsy showed rupture of spleen, liver and left kidney.
2. Walker. Laparotomy: small bowel withdrawn from rent, which it was impossible to suture. Recovered.
3. Knaggs. Laparotomy by Berry, 5 days after injury; hernia reduced; liver sutured against rent in diaphragm. Died on table.
4. Martin. Operation for incarceration 5 days after injury. Died.
5. Suter. Incision as for nephrectomy; suture of rents in diaphragm, and left nephrectomy. Recovered.

McGuire (1914) has reported a successful operation for diaphragmatic hernia seven months after rupture of the diaphragm sustained in a fall from a height of 34 feet.

(D) **Operative Wounds of the Diaphragm.**—Iselin (1907) refers to four instances of operative wounds of the diaphragm, recorded by Humbert, Leisrink, König, and Hahn. The operations of Humbert, Leisrink and König consisted in resecting part of the diaphragm for sarcoma; that of Hahn was for enchondroma. Humbert and König's patients recovered, but those of Leisrink and Hahn died.

II. Injuries of the Stomach.—From its anatomical relations, surrounded by liver, diaphragm, pancreas, colon, and spleen, it is very rare to encounter uncomplicated wounds of the stomach (Fig. 71). Among 228 cases of penetrating wounds of the abdomen collected by Seigel in 1898, the various viscera were injured as follows:

Viscus injured	Stabwounds	Gunshot wounds
Stomach	4	71
Small intestine	12	52
Liver	6	31
Colon	5	15
Spleen and kidneys	2	14
Omentum, mesentery, etc.	3	13
	32	196

Of 532 cases treated without operative intervention, 238 recovered, and 294 died, a mortality of 55.2 per cent. Of 376 cases in which operation was done, 182 recovered and 194 died, a mortality of 51.6 per cent. The difference, when the figures are thus given between medical and surgical treatment, is not striking; but when regard is had to the time elapsing between the accident and the operation, it is quite evident that surgery offers the only reasonable hope of cure.

Operations for Penetrating Wounds of Abdomen

Operation	Mortality
Within 4 hours of injury	15.2 per cent.
From 5 to 8 hours after injury	44.4 per cent.
From 9 to 12 hours after injury	63.6 per cent.
More than 12 hours after injury	70.0 per cent.

(A) **Stab-wounds.**—The stomach is one of the organs least often wounded in penetrating stabwounds through the abdominal wall. Among 75 instances of penetrating wounds of the stomach collected by Siegel, there were only 4 cases of stabwound. In former times bayonette wounds of the stomach were not very rare, and Tuffier (1907) called attention to the fact that suicidal stabwounds are n cause, especially in women, who, in aiming at the supposed locatioa of their heart (below the left breast), usually wound the fundus of the stomach.

The *diagnosis* depends more upon the symptoms than upon the position of the wound. In rare cases the stomach may be penetrated by a stabwound in the back (Benoit). As previously noted, wounds of the stomach are not infrequent complications of transpleural per-

forations of the diaphragm (p. 284). Shock, vomiting of blood, and the escape of gastric contents through the wound may render the diagnosis of perforation of the upper intestinal tract certain; but in all cases the wound should be explored before peritonitis has had time to develope, for the question as to which viscus is injured is of relatively little importance. In Lyng's case (1904) there was no doubt as to the penetration of the stomach, since the instrument (a hayfork) which produced the wound, had pieces of meat and potato sticking to it, and the patient had just finished a meal composed largely of these articles.

Treatment.—If protrusion of the omentum or other abdominal contents renders the fact of penetration of the abdomen certain, no hesitancy need be felt in freely opening the peritoneal cavity; but if the stab-wound is small and there is doubt as to whether the blade has actually entered the peritoneal cavity cautious exploration should be undertaken. The patient being anesthetized, and prepared as for an abdominal operation, the surgeon should dissect down layer by layer, and thus follow the track of the wound. It is sometimes impossible to follow a small stab-wound directly; under these circumstances it is best to lay bare the abdominal aponeurosis (sheath of the rectus, aponeurosis of external oblique) over a wide area, and search it carefully for the stabwound. If this cannot be found, and it is known that the blade was very short (that of a pen-knife for example), and if there are no other symptoms of penetration, the skin incision may now be closed. If, however, it be ascertained that the blade has penetrated the aponeurosis, the surgeon should next lay bare the transversalis fascia and peritoneum, but should not open the latter until he is sure it has been penetrated. It is often impossible to trace a small stabwound across fatty tissue and muscular fibres; and it is usually inadvisable to make any attempt to insert a probe in the path of the wound, as by doing so not only are false passages usually produced, but the probe itself may penetrate the abdominal cavity when the latter had not been invaded by the original instrument. But by

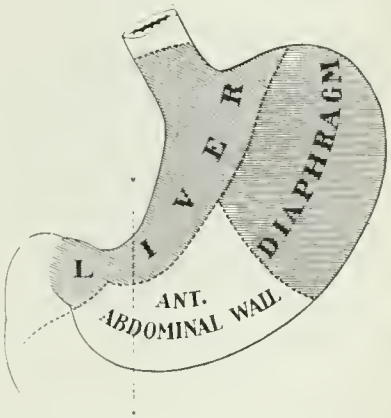


FIG. 71.—Diagram to Show Overlapping of Anterior Gastric Wall by Surrounding Structures.

arresting his dissection at the aponeurotic and peritoneal layers of the abdominal wall, the surgeon will be able to determine in almost all cases the existence or the absence of penetration of the abdominal cavity. We are thus insistent upon this cautious approach, and upon determining beforehand whether the peritoneal cavity has been penetrated by the weapon, because it often happens that on opening the abdomen widely in these cases inspection shows no evidence of intra-abdominal lesion, and very extensive search becomes necessary to exclude the possibility of visceral injury; and if none be found to exist, and it is shown that the vulnerating weapon itself had never opened

Fig. 72.—Diagram to Show Relations of Posterior Gastric Wall to Surrounding Structures.

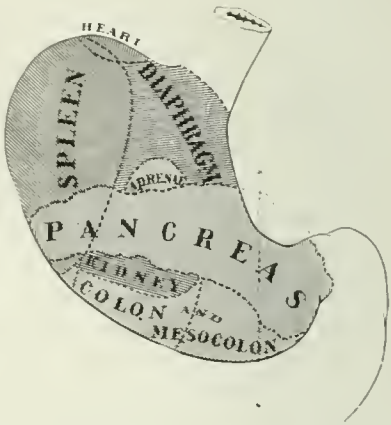


Fig. 73.—Diagram Made by Superimposing Fig. 71 on Fig. 72 to Show Close Relations of Stomach to Surrounding Structures.

the peritoneal cavity, the surgeon will have subjected his patient to a quite unnecessary and by no means trivial operation. If, however, the fact of abdominal penetration has been definitely determined in doubtful cases by the method just described, the surgeon will be quite justified in his extensive intra-abdominal manipulations, even though no lesion be found more serious than hemorrhage from an omental vein.

Although the mortality for penetrating stabwounds of the abdomen is in general about 50 per cent., yet the earlier the operation, the greater the chance of recovery. Siegel found that for those patients operated on within the first twenty-four hours, the mortality was only 8.7 per cent., considerably less than for gunshot wounds. In addition to the 9 operations for isolated stabwound of the stomach tabulated in the first edition of this work, we have found notes of a tenth such

operation, by the late Prof. John Ashhurst, Jr., at the Pennsylvania Hospital, in 1897; his patient did not recover, so of the ten recorded operations, the mortality was 20 per cent.

Stab-wound of Right Gastro-epiploic Artery.—Maiocchi reported in 1908 an operation for a large hematoma in the gastro-colic omentum due to a stabwound of the right gastro-epiploic artery, without other important lesion.

(B) Gunshot Wounds of the Stomach.—In only 32 out of 126 cases of gunshot injuries of the stomach, collected by Forgue and Jeanbrau (1903), was this the only viscus wounded; in other words, in about three out of every four cases gunshot wounds of the stomach are complicated by serious injuries of neighboring organs. The position of the stomach, moreover, is such that not only will it be liable to injury in the case of gunshot wounds of the abdomen, but also in those of the lower thorax, its greater tuberosity reaching as high as the fifth rib on the left. This is one of the reasons why gunshot wounds of the lower thorax give a higher mortality, and more urgently call for operative treatment than do those of the upper portions.

Gunshot injuries of the stomach are divided by systematic writers into: (1) *Perforations*—usually double, there being one wound of entrance, and another of exit; (2) *Abrasions*, or *Excoriations*—in which the gastric wall is wounded without being penetrated; and (3) *Contusions*—which may affect either the serous or the mucous aspect of the organ.

The occurrence of only one perforation in the stomach may be explained in various ways. Among 112 cases studied by Forgue and Jeanbrau (to whose excellent monograph, already quoted, surgeons are indebted for most of their modern statistics), in only 13 did no more than one perforation exist. The ball may never have entered the stomach at all, having merely struck it a glancing blow, sufficient to penetrate its cavity, but because of its tangential course passing on without traversing this cavity. In only 3 cases was the bullet known to have lodged in the stomach. It may make its exit by perforating the duodenum near the pylorus, or the esophagus close to the cardia; and it is even conceivable that a second perforation (wound of exit) may occur in the small retroperitoneal portion of the gastric wall below the cardia, and that the second perforation will thus be impossible of discovery from outside the stomach, as the serous covering of the organ will have been wounded only in one place. Cases are on record in which the bullet, entering the stomach, has been passed subsequently by the bowel, and one in which the bullet was vomited.

According to Forgue and Jeanbrau there were on record only 4 cases of the second class of gunshot wounds of the stomach; while contusions, forming the third class, are equally rare. In a patient of their own, the bullet penetrated and lodged in the stomach; it produced a contusion (undiscovered at operation) of the mucous membrane of the opposite wall. Although this patient did well for three days after operation, he died eventually from profuse bleeding into the stomach from an ulcer which formed as a result of this traumatism.

A distinction is to be drawn between gunshot wounds of military, and those of civil life. The former are almost always rectilinear, owing to the high velocity of the projectile; and the bullet as a rule perforates the patient's entire body, unless arrested by bone; or at least lodges beneath the skin on the distal side of the body. Bullets from injuries of civil life, however, are more easily deflected from their course on entering the body, and rarely if ever pass completely through the trunk. The high initial velocity of the modern military bullet gives it also a well defined explosive action on a hollow viscus if filled with fluid or semi-solid matter, provided the range be less than 400 meters; civil bullets, on the contrary, owing to their much lower velocity, scarcely ever have an explosive effect, even on a full stomach. Wounds by shell fragments combine the evil effects of missiles of high and low velocity.

Symptoms.—The most important symptoms are those of shock and internal hemorrhage. Hematemesis is frequent. Operation should be undertaken before evidences of peritonitis have time to develop.

Diagnosis.—Though perforation of the stomach may be suspected in any patient in whom the wound of entrance lies in the area of vulnerability of the stomach (see Fig. 71), and also in certain other cases where the known course of the bullet lies in the direction of the stomach, though the wound of entrance may be at some distance (loin, thorax perineum, etc.); yet the only certainty consists in exploratory laparotomy.

Prognosis.—Apart from the method of treatment adopted, the condition of the stomach when wounded has a most important influence on the prognosis. If the stomach be empty, and the patient remain in the recumbent position after the injury without being transported for a great distance, extravasation will be very limited, and the development of peritonitis much delayed. Forgue and Jeanbrau include in their study 45 patients treated without operation, and 82 patients treated by laparotomy. In the case of the former patients the death rate was 46 per cent.; in the latter it was 42 per cent. Taking

these figures as they stand, they do not seem to encourage surgeons in urging laparotomy as a life-saving measure. But these operations are not of recent date, and it is but reasonable to suppose that the immediate mortality of operations for gunshot wounds of the stomach has improved along with that of all other departments of gastric surgery, since the date—17 years ago—when these studies were published. Accordingly it is no surprise to find that among 25 recently reported operations for gunshot wounds of the stomach, collected in 1907 by Walton Martin of New York, there were only 6 deaths—a mortality of less than 25 per cent. And although it is probable that both series of statistics give too favorable a prognosis, being founded on collected cases, rather than on a consecutive number of cases treated in one hospital, or by one surgeon; yet their comparative value, showing a marked reduction in the mortality after modern operations, is not affected by this circumstance. But even with the less recent figures of Forgue and Jeanbrau, a little closer inspection will show that the difference between the results of expectant and operative treatment is quite conspicuous. If, for example, we consider apart those cases in which the stomach was the only organ injured, and those in which the gastric lesions were complicated by other serious injuries, we find that in the former class of patients (19 cases), operation was attended by a mortality of 42.10 per cent., while among the latter (63 cases) the death rate was 68.25 per cent.; if, however, no operation was employed, 46 per cent. of those patients (13 cases) with only gastric lesions died, and 93 per cent. of those (32 cases) with wounds of other organs succumbed. As it is absolutely impossible to know before beginning the operation whether other organs besides the stomach are injured or not, it is incumbent upon the surgeon to operate on all cases, in order to decrease the mortality of the much more frequent class of injuries from nearly 100 to less than 70 per cent. or, if we accept Martin's more recent figures, to as low as 25 per cent.

In military practice, gunshot wounds of the stomach, as of other portions of the gastro-intestinal tract are almost always fatal. No operation can be undertaken successfully on the field of battle; and unless a Mobile Hospital or Auto-Chir. is established very near the front, well equipped for prompt abdominal surgery, such patients are either already dead, or peritonitis is so far advanced as to render operation unjustifiable by the time the Base Hospital is reached. Among 965 gunshot wounds of the abdomen treated by operation, which were studied by Wallace (1917), there were 82 which involved the stomach: this was the only organ wounded in 55 cases, and among

these the mortality was nearly 53 per cent.; in 27 cases other viscera were wounded as well as the stomach, and in this series of cases the mortality was over 77 per cent.

The importance of early operation in civil life may be seen from the following figures, copied from Forgue and Jeanbrau:

I. Wounds of the Stomach Only	Cases	Recovered	Died	Mortality per cent.
Laparotomy within six hours of injury....	13	9	4	30
Laparotomy after unknown lapse of time.	6	2	4	66
	19	11	8	42

II. Wounds of Other Viscera as Well as the Stomach	Cases	Recovered	Died	Mortality per cent.
Laparotomy during first six hours.....	29	13	16	55
Laparotomy during second six hours.....	13	2	11	85
Laparotomy after lapse of twelve hours...	13	2	11	85
Laparotomy after lapse of unknown time.	8	3	5	62
	63	20	43	68

Treatment.—The wound in the abdominal wall should be disinfected, its margins being excised, and any particles of clothing carried into the wound (as in President McKinley's case) should be removed. Discarding the instruments used for this purpose, the surgeon should open the abdomen by an epigastric incision, passing through the left rectus muscle *close to the median line.* It is proper to follow the track of the missile only when there is reasonable doubt of its penetration. A very oblique impact in a patient with a very fat abdominal wall may result in the missile making a nonpenetrating wound involving only the abdominal wall, with lodgment of the bullet in the flank, the hypogastrium, or even one of the thighs. But in the vast majority of cases there will be no doubt of the bullet's penetration; and under these circumstances a median incision gives the best exposure. The "head high" (reversed Trendelenburg) posture, with a sandbag under the patient's lower dorsal spine, is a great help in exposing the field of operation. Intrabdominal hemorrhage must first be checked; then the search for perforations is begun. The stomach is to be located immediately beneath the left lobe of the liver, and as soon as a perforation is found, it should be wiped clean, and inverted with at least two

rows of Lembert sutures of fine linen thread. The first tier may be in the form of a pursestring suture. It is not advisable to search for other perforations until the first has been sutured. If a wound of exit cannot be found on the anterior wall of the stomach, the gastro-colic omentum should be divided, between clamps, and below (on the colic side of) the gastro-epiploic arteries, for a distance *at least* of 10 cm.; (Fig. 159) or the intercolo-epiploic route (Fig. 11) may be adopted. A free abdominal incision and a free opening in the gastro-colic omentum will do much to hasten the subsequent steps of the operation. The existence of a perforation in the posterior wall usually will be indicated by extravasation within the lesser peritoneal cavity. Baker's diagnostic dose of methylene blue, taken by mouth shortly before operation might prove useful in such cases. (See p. 124.) Walton Martin states, however, that among the cases he studied, the failure to suture the bullet-hole in the posterior wall of the stomach had not materially influenced the mortality. Should, however, a perforation be detected in a position which was inaccessible to suture, the surgeon may by gastrotomy (incising the anterior wall of the stomach) evert the posterior wall through the gastric incision, and suture the posterior perforation from its mucous surface. Sometimes the perforation in the anterior wall of the stomach is not accessible through a median wound. This is most frequently the case when the perforation is at the fundus or near the cardia. Under such circumstances it is best to adopt temporary resection of the costal margin, after the plan advocated by Auvray. In this an incision is made from the upper angle of the median laparotomy wound, at the ensiform process, obliquely downward to the tip of the tenth left rib; this incision is carried down to the costal cartilages, and these are then divided, from their sternal attachment outward, including if necessary the cartilage of the tenth rib itself. If care be exercised to keep the line of incision in the cartilages, and not to invade the osseous structure of the ribs, the pleural cavity will not be opened. By pulling upward on the costal border thus cut loose, the transversalis muscle and the diaphragm may be detached from the internal aspect of the ribs, and then in order to open the peritoneal cavity it only remains to divide the peritoneum and the transversalis fascia (Forgue and Jeanbrau). The little extra time occupied in making this section is fully justified by the free exposure it gives of the fundus of the stomach. Should the diaphragm have been perforated by the bullet, it would probably be better to approach this region of the stomach by the transpleural route (p. 286). Whenever the gunshot wound traverses the thorax and wounds

the abdominal organs the question will arise as to the advisability of draining the pleural cavity. Walton Martin advocated drainage of the pleural cavity in the majority of such cases; but we are inclined to believe it is safe at the present day to close the pleura, and only to establish drainage secondarily if an empyema develops.

After repairing the gastric wounds, search must be made for other perforations, in the colon, the duodenum, and neighboring coils of small intestine. Wounds of the liver and spleen, and sometimes those of the pancreas, owing to the free hemorrhage which usually attends them, will probably have received attention even before those of the stomach.

Usually sufficient drainage of the lesser peritoneal cavity can be obtained by a cigarette drain carried down through the gastro-colic omentum. In rare cases it is advisable to make drainage through the left loin, below, or even above, the tip of the twelfth rib. This route was particularly studied by Mauclaire (1902), and was successfully employed by Hodge (1908). In very few cases will it be safe altogether to dispense with drainage of the lesser peritoneal cavity. This should never be done if there is the least suspicion of injury to the pancreas.

The clamps left on the cut margin of the gastro-colic omentum are now used as tractors, drawing the colon up against the stomach; and by their aid the incision in this structure may be repaired by sutures (which also serve for hemostasis), except where the drain emerges. If the operation is done within a few hours of injury it will not be necessary to leave another drain to the sutured area on the anterior gastric wall; but in late cases this should be done, and in all cases drainage of the pelvis should be established through a suprapubic incision.

If temporary resection of the costal arch have been employed, it will of course be necessary to re-attach the diaphragm to the lower surface of the cartilaginous flap, and to restore the latter to its place by the aid of deep sutures including skin and intercostal muscles.

Should suture of the perforation cause stenosis of the pylorus or duodenum, gastrojejunostomy should be done at the time of the primary operation, as should gastrostomy if obstruction of the cardia was produced.—In desperate cases with wounds which cannot be repaired, the surgeon must rely on a rubber tube passed into the perforation, and cautiously surrounded by gauze packs.

(C) **Rupture of the Stomach.**—This may be either the so-called "*spontaneous*" rupture, or the *traumatic* variety.

(1) Under the name **spontaneous rupture of the stomach** certain cases have been reported, some of which are more accurately described as ruptures from within (pseudo-spontaneous ruptures), since they were clearly due to the trauma inflicted by lavage. Long ago, Orth recorded a fatal gastric hemorrhage from the passage of a stomach tube; and Key Aborg and Strassmann have each observed multiple ruptures of the gastric mucosa, as the result of too forceful lavage in patients dying of opium poisoning. In another patient of Strassmann's, with gastric cancer, complete rupture of the gastric wall was produced by lavage, and at autopsy the stomach contents were found in the peritoneal cavity. In a patient of Wunscheim's a carcinoma of the esophagus ruptured into the aorta after the passage of a sound, and at autopsy there were also found rents in the mucous coat of the stomach. Haberda has reported a fatal case of complete rupture of the stomach due to its artificial distention with air; Ungar observed a similarly fatal case due to the distention of the stomach produced by swallowing effervescent powders; and Bardachzi (1911) recorded another case in which free hemorrhage and tetanoid cramps followed the administration of a Seidlitz powder in two parts. Though recovery ensued in this case, a second patient with gastric carcinoma died in collapse, apparently from interference with the heart's action.

These ruptures from over-distention are more frequent along the lesser curvature, radiating from the cardia. They have been particularly studied by Key Aborg (1891) and by Fraenckel (1907), each of whom conducted experiments to test the elasticity of the stomach, and its most frequent site of rupture. With a view to preventing rupture, it is above all things important to perform lavage with gentleness, and at leisure; especially is this the case with patients whose stomachs are known to be seriously diseased, and in those who are unconscious, since the sensations of the patient form a very valuable guide to the quantity of fluid which may safely be introduced into the stomach.

True spontaneous ruptures appear to have been recorded in about a dozen instances. Abstracts of these cases follow, the first seven references being quoted from the well known paper on injuries of the stomach by Petry, 1896:

1. Brush. Sudden pain and collapse during effort to lift a stone; slow recovery without operation. For forty years suffered from gastric troubles. At autopsy there was found a gastro-pancreatico-duodenal fistula; the pylorus was tightly stenosed.

This case appears rather apocryphal in the light of modern knowledge.

2. Newman. Insane patient; during violent vomiting after meal, developed pain, collapse, tympany, subcutaneous emphysema. Rupture of entire gastric wall found at autopsy.
3. Revilliod. Spontaneous rupture from fermentative distention. Autopsy showed no ulcer; two ruptures in serous coat.
4. Hoffman. Spontaneous rupture from fermentative distention. Death.
5. Thompson. Spontaneous rupture from unknown cause. No injury.
6. Lantschner. Stomach in umbilical hernia. After drinking immense quantity of water and tea, rupture caused by vomiting. Died.
7. Chiari. Spontaneous rupture from fermentative distention. Autopsy showed longitudinal rupture in scar of old ulcer.
8. Hartmann (1906). Spontaneous rupture; autopsy showed extreme atrophy of gastric walls. (Perhaps auto-digestion.)
9. Ipsen (1907). Spontaneous rupture from vomiting. Death.
10. Wilke (1907). Autopsy on patient with volvulus of stomach, showed rupture of its posterior wall causing death from peritonitis.
11. Ambrose (1908). Spontaneous rupture while stooping. Operation 1½ hours later showed a slit 1 in. long near pylorus on margins of chronic ulcer. Partial gastrectomy. Recovery.
12. Gill (1918). Girl aged 16 years, with healed kyphos (tuberculous). Death in collapse after illness of few hours duration, with symptoms of acute dilatation of stomach. Autopsy showed a complete rupture 2.5 cm. long on anterior wall near greater curvature, 5 cm. below cardiac end, with radiating rents in the mucosa.

As pointed out by Doujon (1903), it is quite probable that some form of gastric volvulus, self reduced before death, or undiscovered at autopsy, is the chief cause of such spontaneous ruptures in apparently healthy stomachs; and the case since reported by Wilke, and above quoted, supports this view. Strassmann (1907) suggested that these mucous lacerations may be much more frequent than is generally appreciated, and questioned whether they might not form the initial stage of gastric ulcer or carcinoma. As already mentioned, other writers seem to have had the same idea. (See p. 245.)

According to Rehn (1896), spontaneous rupture from gaseous dis-

tention is not very rare in horses, but generally is observed along the *greater* curvature.

Gastric hemorrhages, possibly due to ruptures of the mucosa, have been observed in patients who have fallen on the back, on the buttocks, and even in one injured by a "general shaking up." (Strassmann, loc. cit., S. 166.)

(2) **Traumatic Rupture of the Stomach.**—This is usually accompanied by such extensive visceral injuries as to terminate fatally before surgical treatment can be instituted. Geill in eight and a half years found 35 ruptures of the stomach at autopsy in Vienna, and Strassmann observed about the same number at autopsies in Berlin, during about fifteen years. Two such cases have been observed at the Episcopal Hospital, Philadelphia (1901 and 1910): in the first case the lesion was found at autopsy; but in the second case operation was done (by the junior author), but the patient died within an hour. As an injury which interests surgeons it must be considered rare.

The causes are blows, falls, and crushes, especially the last. Kicks by horses are also a frequent cause.

Rehn (1896) divided ruptures of the stomach into (1) those involving only the serous coat; (2) those affecting the serous and muscular coats; (3) interstitial ruptures, including submucous hematomata, etc.; (4) ruptures of the mucosa; (5) penetrating ruptures—those in which the entire thickness of the gastric wall is involved. From the experiments conducted by Fraenckel and others, it appears that mucous ruptures are usually produced by overdistention from within; that ruptures of the serous coat alone are frequently the result of external pressure on a distended stomach; that contusions result in interstitial ruptures; and that complete ruptures are more apt to be caused by crushing of the stomach against the spinal column.

Petry (1896) in his study of subcutaneous ruptures of the alimentary canal, found the stomach involved in 21 cases, the small bowel in 172 (of which 9 were duodenal ruptures), and the large bowel in 26 cases. Of the 2 gastric ruptures, eight he termed spontaneous, including here the case reported by Key Aboig (which we have classed as pseudo-spontaneous). These cases have already been discussed (p. 297). Of the 13 traumatic ruptures, two were submitted to operation; death quickly ensued in five of the remaining patients (11 in number), on whom no operation was done. Of the six patients, who survived without operation, two recovered with gastro-cutaneous fistulæ, two were operated on after 4 and 2 weeks respectively, for perigastric abscesses, but eventually recovered; while in the remaining two pa-

tients, traumatic ulcers formed, which subsequently necessitated further treatment. Tawastsjerna collected, in 1905, 125 cases of operation for subcutaneous rupture of the abdominal viscera, all that had been recorded since the publication of the paper by Petry, mentioned above. Of these more recent cases, 41 recovered, and 84 died, a death rate of 67.2 per cent. Meerwein in 1907 studied 93 operations for subcutaneous rupture of the abdominal viscera, recorded since 1896. He found that among 69 patients operated on within 24 hours of the injury, 38 died, a mortality of 55.1 per cent.; while of 24 patients who were not operated on until more than 24 hours had elapsed, no less than 14 died, a mortality of 66.7 per cent. Although these figures probably are relatively correct, in that they show the value of early operation as a life saving measure, there is also no doubt that, as Meerwein points out, these results are much more favorable than are actually obtained in any consecutive series of cases. To support this assertion, Meerwein quotes the following series of operations for rupture of the abdominal viscera.

Authority	No. of operations	Recovered	Died	Mortality per cent.
Neumann	21	1	20	95.2
Schmitt	7	1	6	85.7
Voswinckel	14	2	12	85.7
Hagen	17	1	16	94.1
Thommen	17	2	15	88.2
Basel clinique	18	3	15	83.3
Hildebrand	12	5	7	58.3
	106	15	91	85.8

It should be noted that the cases reported by Hildebrand have all been treated within recent years.

Only eight operations for traumatic rupture of the stomach appear to have been recorded. The patient of Mikulicz (1885) had been drinking heavily, and was taken with sudden abdominal pain, followed by extreme tympany and collapse. Mikulicz first punctured the abdomen; the gas which escaped was inflammable, and smelled strongly of alcohol. By laparotomy a rupture of the lesser curvature was sutured, but the patient died three hours later. Although the stomach evidently was diseased prior to the time of rupture, it was the opinion of Mikulicz that the rupture was traumatic in origin, due either to distention from within the stomach or to unrecorded external injury.

Rehn's patient (1896) fell from a second story window, but recovered after prompt suture of the rent in the stomach. The patients operated on by Thommen and by Körte both died; but Thommen's patient (1902) lived four days, and death was found at autopsy to be due to rupture of the duodenum undiscovered when the rupture of the stomach was sutured. Allivasato's (1909) and Rodocanache's (1911) patients recovered; but Singley's (1913) died in 7 hours. The history of Ashhurst's patient (already mentioned at p. 299) is as follows:

Charles B., 7 years of age, was brought by ambulance to the Episcopal Hospital, at 1.30 P.M., Nov. 16, 1910. Thirty minutes previously, soon after his midday meal, he had been knocked down by a heavy wagon, the wheels passing across his epigastrium. He vomited a little blood, and was in a state of shock; his temperature was subnormal, his pulse rapid and fluttering; and his lips were blue. The abdomen was a little distended and tympanitic; and anteriorly the liver dulness was replaced by tympany. There was dulness, however, in the right flank. There was much abdominal pain, but no rigidity. One hour later it was noted that the pulse rate was 140, that the child was thirsty, and that there was sighing respiration. A diagnosis was made of internal hemorrhage, probably accompanied by rupture of a hollow viscus.

Operation (Dr. Ashhurst), 2 hours after injury (Ether): Paramedian epigastric incision from ensiform to navel; liquid blood and particles food (chiefly boiled potato) poured forth. Packs were inserted to the left and below, exposing the pylorus. Blood poured from above the stomach. The gastrohepatic omentum was ruptured, and about 100 cc. blood clots and an equal amount of food lay between the stomach and the left lobe of the liver, in the lesser peritoneal cavity. On the posterior wall of the stomach near the lesser curvature was a large circular rupture (about 4 cm. in diameter), with the mucous membrane everted into the opening. The patient's lower dorsal spine was now raised on a support, as in operations on the biliary tract, to secure better exposure. The rupture in the stomach was closed with two layers of sero-serous sutures. The continuous ooze of blood from the structures beneath the peritoneum forming the posterior wall of the lesser peritoneal cavity, and from several small ruptures on the under surface of the left lobe of the liver, was controlled by three gauze packs, emerging through the gastrohepatic omentum. The epigastric incision was closed with through-and-through sutures of silkworm gut, and a glass tube was placed through a suprapubic stab-wound, to drain the pelvis. Pieces of boiled potato floated out of this tube. During the operation, which lasted one hour, the patient received 1500 cc. of saline solution, with adrenalin, intravenously.

Death occurred a little more than an hour after his return to bed.

Interstitial ruptures of the stomach may cause (1) Hematoma; (2) Cyst; (3) Abscess. This subject has been made the subject of an interesting monograph by Pedro Chutro, of Buenos Aires, and it is from his work (1905) that most of what follows has been abstracted. Rupture of a vessel in the submucosa is possibly the first lesion; and the hematoma which forms may be gradually absorbed, without producing very acute symptoms. If of large size, however. a cyst will form, and usually this cyst becomes more or less infected from transudation through the mucous membrane which forms one of its walls (p. 214). A certain amount of gastric juice may be secreted directly

into the cyst, from the deep layer of the mucosa. By some such process as this there is formed an abscess, which in the patient on whom Chutro operated resembled a cold abscess in character. Zeigler operated on a patient who had received an abdominal injury some weeks previously; he found a large hematoma in the anterior wall of the stomach; recovery was uneventful. Similar cases, without, however a history of injury, have been recorded by Sloane and Anderson. Sloane's patient died from typhoid fever, and the gastric condition was not suspected during life. Anderson's patient died after the hematoma had been drained by laparotomy. Rendu operated on a patient with an infected cyst of the gastric wall; death occurred from peritonitis. Gallois, Houlong and Leflaive recorded a case where death was due to rupture of a cyst of the posterior wall of the stomach. Chutro's own patient, a boy of 9 years, received a kick from a horse in the epigastric region; 19 days later an interstitial abscess in the posterior gastric wall was opened and drained by laparatomy; uneventful recovery ensued. Although a history of injury is certain only in the patients of Zeigler and Chutro, it seem proper to mention the others in this place, in view of what we have already learned of the character of mucous and spontaneous ruptures of the stomach.

Menne (1905) made an extensive study of the **after effects of injuries of the stomach.** He collected 51 cases in which lesions of the gastric mucosa (ulcer, stenosis, etc.) followed trauma, 45 being due to direct, and 5 to indirect violence, while in one case the nature of the injury was not stated. In 24 cases the injury was produced by moving bodies impinging upon the patient's abdomen, and in 27 cases it was caused by the collision of the patient with bodies at rest. He further tabulates 8 cases of gastric hemorrhage or perforation due to more or less indirect injuries, such as muscular efforts, etc.

Rupture of Right Gastro-epiploic Artery.—An isolated case of this nature, accompanied by an insignificant tear in the margin of the liver, due to the kick of a horse, has been recorded by Vatter (1904). Laparotomy was done on account of symptoms of internal hemorrhage; the artery was ligated, and recovery ensued. One case of rupture of the gastro colic omentum and one of rupture of the gastro-hepatic omentum have been operated on at the Episcopal Hospital by the junior author. The *first patient* (1909), a man aged 64 years, came to operation 23 hours after being struck in the epigastrium by the end of a swinging cable. On opening the abdomen immense quantities of blood were found, and the patient, whose pulse stopped and who ceased to breathe, was only revived by direct massage of the

heart through the abdominal wound. The only lesion found was a rupture of the gastro-colic omentum still oozing blood. The patient had an irreducible left scrotal epiplocele, which, holding the omentum taut, evidently predisposed to injury. The patient died 3 hours after the end of the operation. In the *second case* (1912), a boy 4 years of age who came to operation 2½ hours after injury, there was a rupture of the spleen in addition to rupture of the gastro-hepatic omentum. The patient died with symptoms of sepsis 55 hours after operation; autopsy, however, showed no evidences of peritonitis nor of further intraperitoneal hemorrhage.

(D) **Foreign Bodies in the Stomach.**—Children, insane people, and mountebanks form the chief classes of patients to be considered here. Others by mistake occasionally swallow a tooth-plate, a piece of bone, or some similar object. Children most frequently swallow coins, buttons, pins, etc. The insane swallow almost everything; hair swallowing is done only by those with neurotic taint, frequently during convalescence from some debilitating disease, such as typhoid fever. Sword swallowers occasionally try to exhibit their art with too short a knife; it may slip from their grasp and be hurried into the stomach by involuntary efforts at swallowing. Pieces of glass, nails, and all manner of articles are sometimes swallowed by showmen.

The majority of such articles as pass through the straits of the esophagus and reach the stomach without difficulty will also pass the pylorus, traverse the intestinal tract, and be discharged from the anus in the course of a few days without producing any symptoms. The form, consistency, and bulk of the article swallowed, are, in the order mentioned, the characters which have most influence on the prognosis. Bodies of rounded form, such as coins, or at least those which have no prongs or sharp angles, will usually pass without trouble. Certain other articles, such as pieces of bone, may become partially digested in the stomach, and may thus, with their sharp angles rounded off, be discharged without causing injury to the gastro-intestinal tract. As far as the stomach is concerned, the size of the body swallowed is the least important factor in the prognosis, as it is almost certain that articles which can pass the cardiac orifice can also pass the pylorus. This remark, however, does not apply to long nails, slate-pencils, knives and forks, in which form is of greater importance than mere bulk.

Occasionally, as in children and in the insane, no history of swallowing a foreign body can be obtained. Apart from such a history, the symptoms of the lodgment of a foreign body in the stomach are chiefly those of pain and vomiting. The latter may be persistent, and

the vomitus may be streaked with blood. In other cases, in spite of pain, no nausea is present, and appetite may even be preserved.

The *diagnosis* in acute cases is rarely difficult, because of the history. But it is frequently difficult to determine the presence of hair tumors (Egagropile, Trichobezoar) before operation, because the patient either is not aware that she has been in the habit of swallowing her hair, or she is unwilling to acknowledge the habit. In the insane the diagnosis may be impossible without the aid of the Roentgen rays. The subject of hair-balls in the stomach was studied by Matas in 1915: he collected 73 cases; 44 of the patients came to operation. Among 28 operations for hair-balls in the stomach analyzed by Moore (1914), 2 patients died, a mortality of 7 per cent.

If the foreign body remain in the stomach, it may be quiescent for long periods; it may, on the other hand, cause ulceration, perigastritis, subacute perforation, and finally a subcutaneous abscess; in rare instances it may directly perforate the gastric wall.

The *treatment* to be adopted depends on the nature of the body swallowed. If certain to pass, the Vienna treatment, advocated long ago by Billroth, should be employed. This consists of a diet of mashed potatoes, or such similar substances as will tend to coat the foreign body and aid its passage through the intestinal tract. When once out of the stomach, it is most apt to be arrested at some point in the lower ileum. Under no circumstances should a purge be given. The violent peristalsis thus aroused is much more likely to cause perforation of the stomach or bowel, or intestinal obstruction from inflammatory edema, than to promote the passage of the foreign body through the intestinal tract. If no symptoms are produced by the swallowed article, there need be no haste in resorting to operation, even if it is manifestly impossible for the foreign body to escape from the stomach. The Roentgen rays may be employed, and the location of the offending substance determined. As its weight may cause the stomach to descend much below its normal position, it may appear that the foreign body is in the large bowel (especially the cecum) instead of in the stomach. The passage of a stomach tube, or the introduction of bismuth emulsion into the stomach just before a second skiagraph is made, probably will determine the question.

Gastrotomy is indicated (1) when it is clearly impossible for a quiescent foreign body to be discharged spontaneously; (2) when any symptoms arise from any variety of foreign body; (3) it is occasionally required for the removal of a foreign body impacted in the lower end of the esophagus. The use of endogastric instruments, as employed by

Chevalier Jackson, is justifiable only in the hands of a specialist. The average surgeon will consult his patient's safety much more by resorting to gastrotomy.

This operation, for this purpose, is said to have been first done by Daniel Schwabe in 1635. His patient, operated on without an anesthetic, recovered. It was not until 1848 that the operation was repeated, by Tilanus. In 1887 Bernays collected 11 cases of gastrotomy for foreign bodies, including one of his own; he also referred to 16 other operations which consisted in extracting foreign bodies from the stomach after this viscus had become adherent to the parietal peritoneum as the result of perigastritis set up by the foreign body. Of the 11 patients in the former series, only 2 died. The late Prof. Ashhurst (1893) referred to 50 cases of gastrotomy for the extraction of foreign bodies, 42 of which terminated in recovery. He says that "foreign bodies which have been swallowed, and having ulcerated through the walls of the stomach, had lodged in various parts of the abdominal cavity, have been successfully removed by LeDentu, Bardeleben (two cases), Nussbaum (two cases), LeFillier, and Dubois."

Among 20 recent cases of gastrotomy for foreign body, references to which were given in our first edition, there was only 1 death, a mortality of 5 per cent; and among 23 operations reported since that date there was only 1 death,[1] so that the operation may be considered reasonably safe. Two patients (professional "sword-swallowers") whose cases are recorded by Revenstorf (1904) and by Warbasse (1904), were operated on twice both times successfully. Winslow (1919) has reported a successful case of gastrotomy with removal 1290 different foreign bodies.

The *operation* consists (1) in opening the abdomen through the left rectus muscle, (2) in locating the stomach; (3) in drawing the stomach into the wound, and isolating it by gauze packs; (4) opening the stomach, preferably by an incision transverse to its long axis; (5) removing the foreign body by forceps or fingers; (6) suturing the gastric incision with at least two rows of Lembert sutures, or one of the Czerny and one of the Lembert type; (7) closing the abdominal wound.

The incision in the stomach should be no longer than is absolutely requisite for the extraction of the foreign body. It is well to locate the body and fix it against a convenient portion of the gastric wall before opening the stomach. Should it be impossible to remove a body impacted in the lower esophagus, gastrostomy should be performed;

[1] This series of operations includes one patient, reported by Wolff (1913), who had had no less than five separate gastrotomies done at various times.

this procedure was necessary in a patient under Jacobson's care in 1889; unfortunately the patient did not survive more than two days. In a similar case recorded in 1900 by Edmunds a tooth-plate was successfully removed from the lower esophagus by gastrotomy.

III. Injuries of the Duodenum.—Injuries of the first portion of the intestinal tract differ from those of other portions chiefly on account of the situation of the duodenum; not only is it in close relation with other structures of the greatest importance (superior mesenteric vessels, portal vein, pancreas, etc.), but it also is quite firmly fixed on account of its retro-peritoneal position. Wounds of the duodenum, therefore, are more frequently complicated, as well as more difficult to treat, than are those of the jejunum or the ileum.

(**A**) **Stab-wounds** of the duodenum, unaccompanied by more serious lesions, do not appear to have been observed. It is of course conceivable that such an isolated wound might occur, either through the loin, or from in front, grazing the liver and the colon, or even by passing through the gastro-colic omentum, and reaching the transverse duodenum.

(**B**) **Gunshot wounds** of the duodenum, unaccompanied by more serious injuries, have been observed in several instances. According to Cackovic (1903), a gunshot wound of the duodenum was first sutured by Ramsay, in 1885. Harte (1902) reported a patient with a "grooved" wound of the first portion of the duodenum, and perinephric hemorrhage, who recovered after suture of the perforation of the duodenum by laparotomy, and arrest of the hemorrhage by packing the kidney region through the loin. Summers (1904) operated on a patient who had been shot in the right loin. A double perforation of the duodenum was found, also a perforation of the gall bladder. The latter injury, and the anterior perforation of the duodenum were repaired, by sutures, by laparotomy; and the posterior (retro-peritoneal) perforation of the duodenum, and a wound of the kidney, were tamponned through a lumbar incision. Death occurred in three days from "retro-peritoneal phlegmon," not from peritonitis.

The *treatment* of gunshot wounds of the duodenum is difficult because of their deep situation, and the frequency with which retro-peritoneal injuries are overlooked. Modern experience with mobilization of the duodenum will render access to retro-peritoneal lesions of its descending portion less difficult than heretofore. But the uncertainty which always exists as to the efficiency of closure of retro-peritoneal portions of the intestine, makes the prognosis in such cases particularly grave. It usually will be well to drain the sutured area, particu-

larly if it be retro-peritoneal. Drainage should always be employed, preferably through the loin, if a retro-peritoneal perforation is suspected but not definitely located; or if one is located in an inaccessible place. Resection, with end-to-end anastomosis may sometimes be required. In many instances it probably will be safer to close both ends of the duodenum, and restore the continuity of the intestinal canal by some form or forms of lateral anastomosis, as in the case of Meerwein to be quoted below.

(C) Rupture of the Duodenum. Because of its fixed position against the spinal column, rupture of the duodenum is by no means so unusual as might be supposed. Meerwein (1907) collected 64 cases; and he referred to 18 others included in the tables previously published by Jeannel, the original references to which were not accessible to him. According to Cackovic (1903), operation for rupture of the duodenum was first done in 1896 by Herczel. To show the relative frequency with which the duodenum is ruptured, the following figures are quoted from Gage (1902): Duodenum, 10 cases; jejunum, 20 cases; ileum, 42 cases; colon, 6 cases. Meerwein studied the records of 28 operations for this condition: 16 patients recovered, and 12 died, a mortality of 42.85 per cent. But in 6 of the fatal cases the rupture in the duodenum was not found; so that the mortality attending the completed operations is only 27.27 per cent. Of the six fatal cases in which the rupture was found at the time of operation, three patients died at once, two died later from peritonitis, and one patient (Moynihan's) lived in excellent health for 104 days after the operation, and died then from perforation of the intestine by the Murphy button which had been employed at the operation. Sherwood (1906) has recorded a case of rupture of the duodenum, which is not included in Meerwein's statistics. This patient died seven days after suture of the rupture, from gangrene of the injured bowel. We have references to reports of 27 operations for rupture of the duodenum, published since the first edition of this work; 18 of the patients died, a mortality of 66.6 per cent. But if 6 cases in which the rupture was not found, and all terminating fatally, are omitted, the mortality is reduced to 57.1 per cent., which probably is nearer the truth than the mortality recorded in the series of cases collected by Meerwein.

The transverse is more often affected than the descending portion of the duodenum, and the rupture usually occurs more or less transversely to the long axis of the intestinal canal. The bowel may be completely torn across. This is not unusual at or near the duodenojejunal juncture, as in Moynihan's case; the explanation probably being that the greatest strain is felt where the fixed portion ceases and

the movable portion of the bowel commences. The causes are blows (especially kicks from horses), falls and crushes. Perry and Shaw (1893) refer to a case of rupture of the duodenum produced by vomiting; ruptures of the stomach existed in the same patient; none of the lesions were discovered during life.

Suture should be done where this is possible. If doubt exist as to the viability of the injured gut, excision had best be done, difficult as such an operation is. Sometimes end-to-end anastomosis is possible. In Moynihan's patient, referred to above, a boy of 6 years, a few inches of damaged gut were resected, the proximal end of the duodenum was closed, and the distal end (origin of the jejunum) was united to the stomach; as a result the whole of the bile and pancreatic juice passed into the stomach in order to reach the jejunum. As already mentioned, this patient survived in excellent health for 104 days, and died then from perforation of the bowel by the Murphy button which had been used in making the anastomosis. Meerwein found in his patient a complete transverse rupture of the duodenum where it crossed the spinal column. It was impossible to close the rupture by an end-to-end anastomosis. Accordingly the proximal end was closed by a purse-string suture; the distal end was then drawn out from beneath the root of the mesentery to the patient's left, the devitalized portion of the gut was excised, and the remaining (distal) end of the duodenum closed. A lateral anastomosis was then made between the posterior wall of the stomach and the upper jejunum (trans-mesocolic posterior gastro-jejunostomy); and finally a lateral anastomosis was made between the juxta-pyloric portion of the duodenum and the jejunum about 60 cm. (24 inches) from the origin of the latter (anterior ante-colic duodeno-jejunostomy). As a precaution gauze drains were left to all the sutured areas. The patient recovered.

Foreign Bodies in the Duodenum.—At least one instance (English, 1905) of perforation of the duodenum by a foreign body is on record; the patient was saved by operation. In a case recorded by Lucas (1901), a nail which, as shown by skiagraphs, had been lodged for some weeks in the descending duodenum was successfully removed by duodenotomy. Chaput (1907), Dehmel (1910), Batavyai (1913), and Buchanan (1913), have also removed foreign bodies from the duodenum. Crossan (1916) has recorded the history of a patient under the care of the junor author, from whom a fish bone (lying partly in the duodenum and partly in the left lobe of the liver) was removed at operation from the gastro-hepatic omentum. The patient died from suppurative hepatitis which had developed before operation.

CHAPTER XIV

TECHNIQUE OF OPERATIONS ON THE STOMACH AND DUODENUM

Preparation for Operation.—Whenever practicable, it is well for the patient to pass a night or two in the hospital before the day set for operation. In cases of perforation or of hemorrhage, if the surgeon think it proper to operate for the latter as an acute condition, there is of course no time for delay. When feasbile, from 24 to 48 hours should be devoted to putting the gastro-intestinal tract in as good a condition for operation as possible. For at least twenty-four hours before operation only cooked, and therefore sterile, food should be given the patient. The mouth and teeth should be thoroughly cleansed after each meal and at bed-time with an astringent, alkaline, and mildly antiseptic wash. The mouth should be kept as free as possible from any particles of food which may undergo fermentation.

A brisk purge should be administered the day before operation, preferably in the morning. Usually either Epsom salts or castor oil should be chosen, but the preference of the patient may be consulted. If given in the morning of the day before operation, the effect of the purge will wear off during the day, thus allowing the patient to have an undisturbed night preceding the operation. On the morning of the day of operation, an enema should be given to empty the lower bowel.

After the purge has acted, very little food should be given the patient. If any be given, it should consist entirely of such material as will be readily absorbed and will leave little if any residue in the intestinal tract.

The patient may drink freely of sterile water until within a few hours of the operation. The stomach should be empty at the time of operation, but it is only in exceptional cases that it must be emptied by means of the stomach tube. Where there is marked stasis, especially if there are putrefactive changes in the stomach, it is always advisable to empty the organ immediately before operation.

Preparation of the Abdomen.—In the afternoon before the operation, the abdomen is thoroughly washed with green soap and water, special attention being paid to the navel. The entire abdomen is

shaved and again washed with the green soap and water, gauze being used instead of a brush. It is then rinsed with sterile water, rubbed with 60 per cent. alcohol, and, when thoroughly dry, is covered with a sterile towel, held in place by a few turns of bandage, or a binder. Heavy dressings are avoided, particularly in hot weather, as it is undesirable to cause sweating of the underlying skin. This sterile towel is removed after the patient has been placed on the operating table, after anesthetization. The field of operation is then painted with an alcoholic solution of iodin (3 per cent.), or of picric acid (2 per cent.). It is important in using these solutions that the skin shall not have been wet for several hours and that the skin shall not be covered with sheets, etc., after it has been painted with these solutions until they have thoroughly dried. Neglect of these precautions may cause irritation or even blistering of the skin. Moreover, it is important to be sure that the solutions employed are not of greater strength than indicated: if the receptacles in which they are contained are left long uncovered, the strength will be increased imperceptibly by evaporation of the alcohol.

General Considerations on Operative Technique. Anesthetic.—All these patients deserve the services of a *professional anesthetist.* Ether is used unless contraindicated, the so-called "open, drop-method" invariably being used. Ethyl chloride is never used; it is seductive but dangerous. In all seriously ill or cachetic patients nitrous-oxide-and-oxygen anesthesia is preferred to etherization, as taxing the excretory organs less, and minimizing the risk of post-operative pneumonia. A hypodermic injection of morphin (0.010 gramme) and atropin (0.0005 gramme) should be given about an hour before beginning the anesthetic. The patient is placed upon the operating table in the etherizing room, before anesthesia is begun. We believe that this method reduces the amount of anesthetic administered, ensures the patient lying on the table in as nearly normal an attitude as possible, and thus is advantageous in every respect. Neglect to support the normal arch of the lumbar spine by a small pillow is a frequent cause of post-operative back-ache. Ether is discontinued as soon as possible, and occasionally oxygen is administered as the abdominal wound is being sutured so that consciousness begins to return as the dressing is applied. Care is exercised to maintain bodily warmth during the operation, and in the case of very weak patients the table is covered with a hot water bed; and the patient should wear a cotton jacket and have the legs and arms bandaged in the same material, or should wear long stockings of canton flannel. When

the patient is transferred to bed, if he is perspiring freely, the clothing should be changed.

Assistants. — One assistant helps the surgeon during the operation. One nurse threads needles, hands instruments, etc.; another is in charge of the gauze sponges and hands them to the surgeon as required; while a third changes the saline solutions, keeping them constantly clean and hot; and a fourth also keeps account of all pieces of gauze used, reporting from time to time to the nurse in charge of the gauze, who is responsible for the final reckoning of pieces of gauze employed during the operation. The surgeon does all the operating himself; the assistant holds retractors, and so disposes the operative field as to make operating easy.

Instruments.—Very few special instruments are required. Good **retractors** are a necessity. **Rubber-covered clamps** are extremely desirable. Mechanical aids for gastro-intestinal anastomosis, such as the **Murphy button,** are never used except in emergencies, where it is desirable to terminate the operation rapidly, or in positions where the application of sutures is particularly difficult. Thus in doing anterior gastro-jejunostomy, which is reserved for patients with gastric cancer so far advanced as to make even a posterior palliative operation imposible, the Murphy button sometimes is used, so as to keep the abdomen open the very shortest possible time. **Gauze packs** are absolutely requisite to protect the general peritoneal cavity, to keep other abdominal viscera from prolapsing into the wound, and to maintain the vital heat of those structures which are exposed. These packs are about ten inches square, and are made by basting together six to eight layers of gauze. They are wrung out of hot saline solution as required, and are handed to the surgeon hot. It is convenient to have a tape about 10 cm. long sewed to one corner of the pack; this tape is left protruding from the wound and is clamped by a hemostat. If no pack is ever put entirely inside the wound none will be lost inside the abdomen.

Sutures.—Two main types of sutures are used—the Albert (through-and-through) and the Lembert (sero-serous), as shown diagrammatically in Fig. 74. The **Albert suture** is invariably of absorbable material; chromicized or iodized catgut is employed. This suture seldom is used in abdominal surgery except in the performance of anastomoses. It is designed to be hemostatic, and should stay in the tissues long enough not only to make the anastomosis secure against secondary hemorrhage, but to procure firm union between the margins of the stomach or intestine involved. It should not, however, be of

non-absorbable material, since then it may ulcerate out at one place and by hanging as a loop in the lumen of the newly formed channel (Fig. 35) possibly be the cause of obstruction. Or the portions of the suture still embedded in the tissues may be torn out by the drag of the loop which has ulcerated out, and secondary hemorrhage may

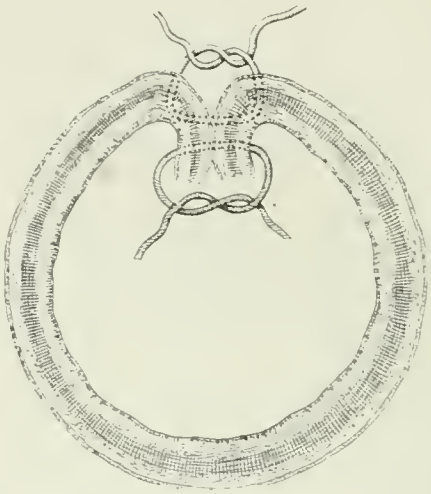


FIG. 74.—Diagram of the Albert (Through-and-through) and the Lembert (Sero-serous) Sutures.

be started or a marginal ulcer formed. These through-and-through sutures are always used as a continuous suture; if there should be danger of the suture puckering the anastomosis, this may easily be prevented by arresting the suture by a knot at three or four points as

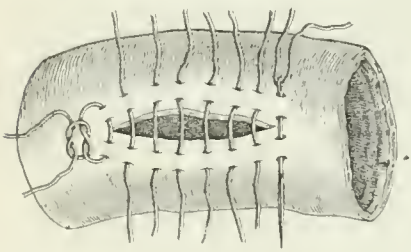


FIG. 75.—Interrupted Sero-serous Suture. (*Lembert's Suture.*)

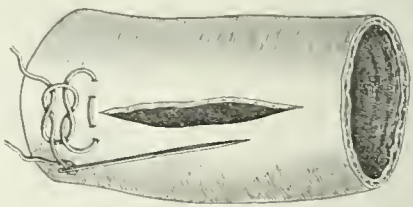


FIG. 76.—Beginning the Continuous Sero-serous Suture. (*Dupuytren's Suture.*)

it passes around the circumference of the anastomosis or by taking a back-stitch one in every eight to ten stitches.

Sero-serous Sutures.—Various forms of this general type are shown in the accompanying illustrations. Linen thread is used in all cases, and the needle picks up all the coats but the mucous. 1. In-

terrupted Lembert Suture (Fig. 75) is especially applicable for reinforcing a continuous Lembert suture at any point where it appears likely to leak. 2. Continuous Lembert Suture, known also as Dupuytren's

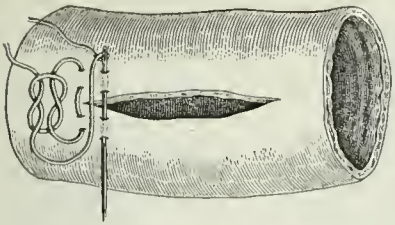


FIG. 77.—Subsequent Steps of the Continuous Sero-serous Suture of Dupuytren.

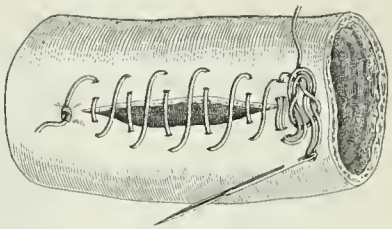


FIG. 78.—Fixing the End of the Continuous Sero-serous Suture by a Knot.

Suture (Fig. 78), is that which is most often used in all forms of intestinal surgery. The suture is commenced by catching up on the needle a bite of the serous, muscular, and submucous coat on each side

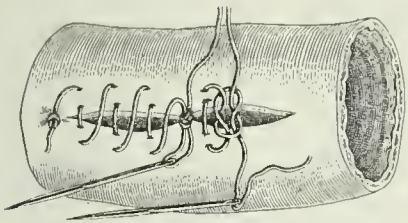


FIG. 79.—Commencing a New Continuous Sero-serous Suture by Tying It to the End of the Previous Stitch.

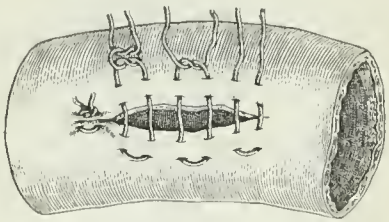


FIG. 80.—Interrupted Sero-serous Suture of the Mattress Type.

of and a little beyond the end of the intestinal wound, the needle being held at right angles to the wound. The suture is fixed at its starting point by tying a square knot (Fig. 76). The needle then again picks

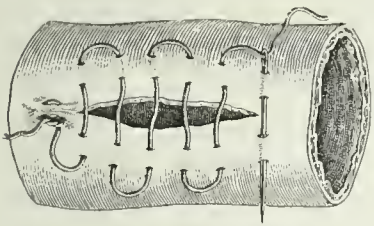


FIG. 81.—Continuous Sero-serous Suture of Mattress Type.

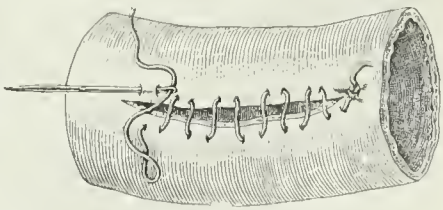


FIG. 82.—Continuous Right-angled Sero-serous Suture of Heyward Cushing.

up all the coats but the mucous on each side of the wound, crossing back to the original side of the wound before commencing each new stitch (Fig. 77), and thus continues until the other end of the wound

is reached, where the thread is knotted as shown in Fig. 78. Should the suture be too short to reach the entire length of the wound, or should it fortunately break, it may be knotted at any point, and a new suture started (Fig. 79). 3. Interrupted Mattress Suture (Fig. 80): this is particularly adapted for places where the intestine is friable, or where there is much tension on the sutures. 4. Continuous Mattress Suture is shown in Fig. 81. 5. Right-angled Suture (Fig. 82), in which the needle is inserted parallel to the edges of the intestinal wound, secures excellent approximation, and is often preferable to the continuous mattress suture, because it can be applied so much more quickly.

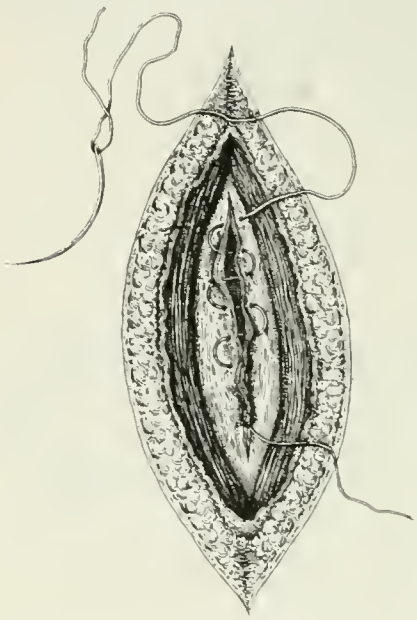


FIG. 83.—Closing the Parietal Peritoneum with Continuous Suture to Evert the Edges, and Bring Serous Surfaces in Apposition.

Closure of the Abdominal Wound.—The peritoneum is sutured by a continuous catgut suture, in such a way that the serous surfaces are everted into the wound, thus bringing serosa against serosa, ensuring rapid union, and leaving no projections within the abdomen to favor adhesions between the scar and omentum or other abdominal viscera (Fig. 83). This suture should begin and end beyond the extremities of the peritoneal wound. Two or three "splint sutures" of silkworm gut are then introduced from the skin surface of one side through all structures but the peritoneum and out again through the other side of the wound (Fig. 84). If the abdominal incision is very short, it is not necessary to use these splint sutures; but in any wound of more than 8 cm. it is safer to employ them. They act not only as tension sutures, relieving the strain on the buried (absorbable) sutures, but they also obliterate all dead spaces between the different layers of the abdominal wall, thus preventing the formation of hematomata and subsequent infection. When these splint sutures have all been placed, but before they are tied, the peritoneal suture first employed is continued downward, as shown in Fig. 85, uniting the anterior sheath of the rectus, and is finally tied to its own

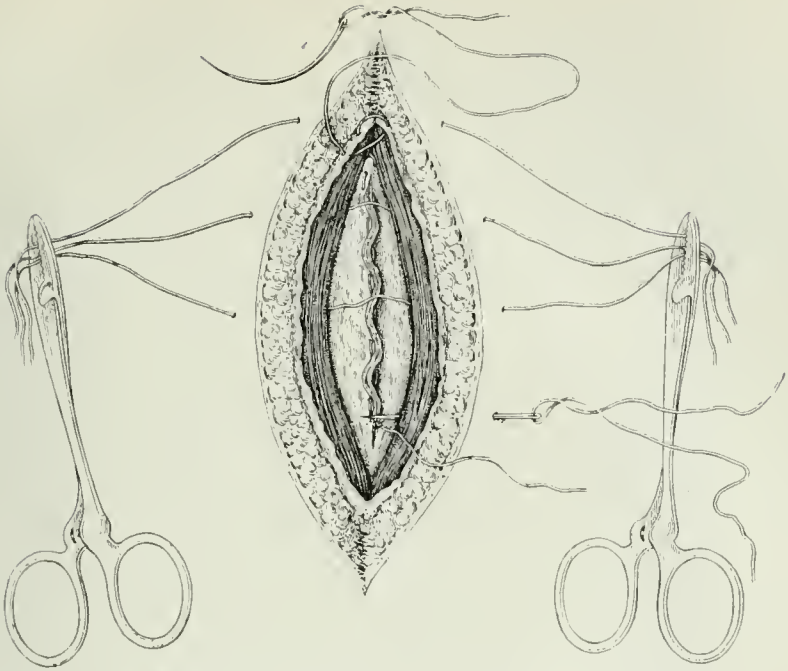


FIG. 84.—Method of Inserting the "Splint Sutures" of Silkworm Gut.

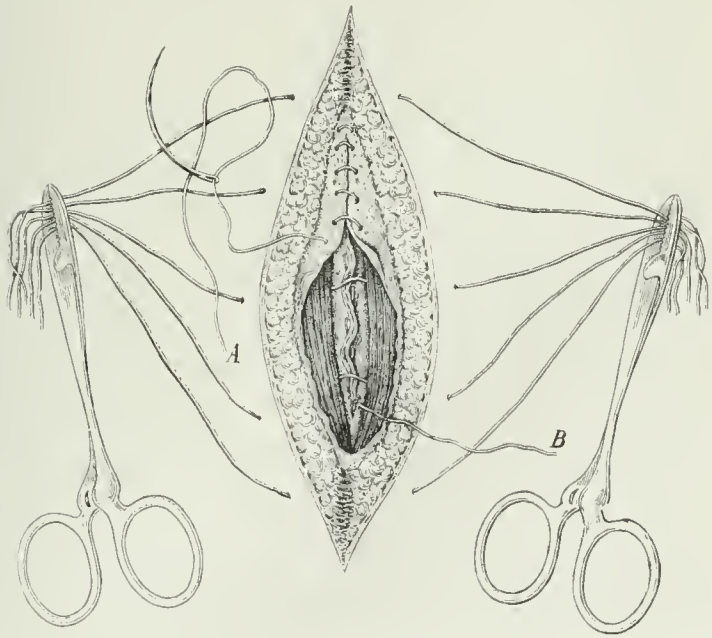


Fig 85.—Suture of the Sheath of the Rectus Muscle.

initial extremity (A to B). Finally the splint sutures are tied (Fig. 86). In suturing an incision in a very obese patient, it is not desirable to close the skin surface too tightly. It is safer to leave space between the sutures for drainage of the fat necrosis.

After-treatment.—The motto for Ward Surgeons is "Let the patient get well." Very little after-treatment except careful nursing is required. A litre of tap water, at a temperature of 105° to 110° F., given by enema before complete recovery from the anesthetic is readily retained; and serves to prevent distressing thirst, to stimulate excretion by the kidneys, and renders very rare the occurrence of retention of urine. The patients are raised up in bed as soon as the effects of the ether pass away; or the head of the bed may be raised about 15 degrees from the horizontal. Vomiting is unusual; it is treated by total abstinence from mouth feeding; by sitting the patient up in bed; by the administration of a glass of hot water and finally by lavage. The patients do not have much pain. If they do suffer from pain, the ward surgeon, after consultation with the chief, is authorized to administer a hypodermic injection of morphin (0.010 gramme) and repeat if necessary. But a minimum quantity of ether, speedy and orderly operating, render the subsequent use of morphin exceptional. Water (at first sips of *hot* water), or small pieces of ice may be given after 12 hours if there is no nausea. Feeding—liquid diet— is not begun for from 48 to 72 hours after operation: at first albumin water, buttermilk, broths, etc., are allowed; but not until flatus is passed or auscultation detects normal peristalsis, is soft diet given. At the end of ten days or two weeks the patients are allowed to get up; but they should not be hurried out of the hospital before their wounds are entirely healed, nor until they are able to take care of themselves.

FIG. 86.—Tying the Splint Sutures.

GASTROTOMY

Indications.—1. For the removal of foreign bodies from the stomach, or from the lower end of the esophagus.

2. As a preliminary to the dilatation of stricture of the pylorus, the cardia or the esophagus.

3. For the control of hemorrhage within the stomach.

4. For the removal of polypi or other pedunculated tumors from the interior of the stomach.

5. As an incident in certain operations on the posterior wall of the stomach.

Incision.—This is to be made through the left rectus muscle close to the median line, from the tip of the ensiform process downward for 8 to 10 cm.

Exploration.—Locate the left lobe of the liver; immediately beneath this is the stomach. While the assistant raises the margins of the abdominal incision with retractors, inspect the anterior gastric wall. If the colon bulges into the wound, pack in gauze pads until it stays out of the operative field. If the operation is for the removal of a foreign body, palpate the stomach gently, and try to locate the body to be extracted. When the foreign body has been fixed with the fingers in contact with the anterior gastric wall, other gauze pads should be introduced so as to isolate completely the portion of the stomach wall to be opened. The stomach may be grasped with rat-tooth or Allis forceps, to facilitate this part of the operation.

Opening the Stomach.—When the stomach has been isolated thus a small incision may be made in its anterior wall with a scalpel. If the object is to remove a foreign body, no longer an incision should be made than is absolutely necessary to extract the foreign body; and under these circumstances the incision is best made transverse to the long axis of the stomach, parallel with the gastric blood vessels. If, however, the stomach must be more widely opened, as for exploration of the esophagus or the removal of an endogastric polyp, the incision in its wall is best made longitudinally, and any bleeding points should be caught in hemostatic forceps, which will then serve the useful purpose of retractors. For exploring the esophagus the incision should be made beneath the cardiac orifice, while if the pylorus is to be dilated, or a pyloric polyp removed, the surgeon will naturally place his incision nearer to it. In exploring the esophagus it is well to bear in mind that the lower end of the esophagus turns toward the patient's left, and that the cardiac is frequently more or less obscured by a fold of mucous membrane. Nine times out of ten the inexperienced operator will vainly endeavor to poke a hole through the fundus of the stomach, pointing his finger to the patient's head, instead of obliquely to his right.

Closing the Stomach.—When the endogastric manipulations have been concluded, the stomach wall is to be sutured with at least two layers or sutures (Albert-Lembert). The stomach is then allowed to fall back into the abdomen; the gauze packs are removed; and the abdominal wound closed in the usual way without drainage.

Gastrostomy

Indications.—1. Impermeable stricture of the esophagus, or malignant obstruction of the cardiac orifice of the stomach.

2. A modified form of gastrostomy may be necessary in cases of phlegmonous gastritis.

The operation of gastrostomy, according to Sencert (1905), was first suggested as a remedy for stricture of the esophagus by Engelbert, a Norwegian, in 1837. It was first performed in 1849, by Sedillot, of Strasbourg. In most cases in which it is adopted it is desirable to establish a more or less permanent opening for the purpose of introducing food into the stomach. But in addition to the permanency of the fistula, it is extremely desirable to have a continent opening, one which will not leak; for leakage will not only deprive the patient of the benefit of the food which has been introduced, but will keep his clothing constantly wet between feedings, by allowing the escape of the gastric juice. A third desideratum, much less important, however, than those just mentioned, is that the fistula shall close spontaneously when it is no longer needed.

Among the many methods which have been devised for the performance of gastrostomy, it is our intention to describe only the following: 1. The methods of Witzel, of Stamm, and of Kader, all of which are based on the principle of inverting the gastric wall so as to form a funnel-like channel from the cavity of the stomach to the wall of the abdomen. 2. The method of Tavel, in which a segment of the jejunum is used as the fistulous tract between the stomach and the skin. 3. The methods of Roux, Herzen, Jianu and Willy Meyer, in which an attempt is made to construct a new subcutaneous esophagus, in front of the sternum, by transplantation of portions of the jejunum or stomach itself.

1. **Methods of Witzel, Stamm, and Kader. Incision.**—The incision is made through the middle of the left rectus muscle, from a little below the costal margin downward for about 7.5 cm. In very young children the large size of the liver makes a lower incision preferable. If the interior of the stomach is to be explored, as in cases of stricture of the

esophagus, or for other reason, the operation of gastrotomy, as described at page 317, will first be performed; and then the incision in the anterior gastric wall should be closed except at one end, where an opening should be left just large enough to admit a large rubber catheter (Nos. 26 to 30 French) or drainage tube. The gastrostomy opening should be made about midway between the greater and lesser curvatures, and in the pyloric portion of the stomach for the same reasons that make one select this portion of the stomach (when available) for gastro-jejunostomy, namely its thicker walls, and its function as part of the *canalis gastricus*.

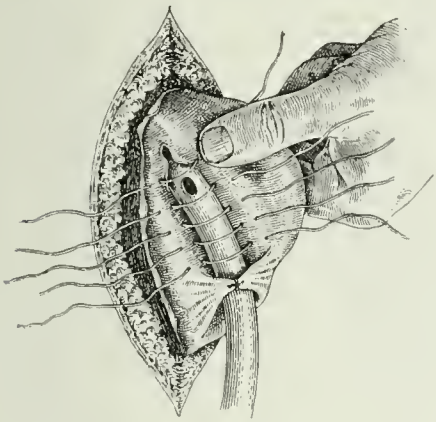


FIG. 87.—Witzel's Gastrostomy. Suturing the Catheter in Place.

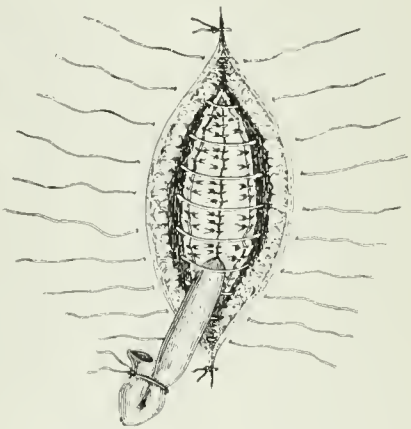


FIG. 88.—Witzel's Gastrostomy. Closure of the Abdominal Wound.

(*a*) **Witzel's Method** (1891).—The catheter is laid on the anterior wall of the stomach with its gastric end toward the lesser curvature, and the stomach wall is then sutured over it by a row of sero-serous sutures (interrupted). When the catheter is thus fixed in a serous channel, a small opening is made in the gastric wall at the extremity of the infolded area, the gastric end of the catheter is passed through this into the stomach for 2 or 3 cm., and is anchored in place by a single catgut suture passing through the catheter and through the entire thickness of the gastric wall (Fig. 87). The opening in the stomach wall is then buried by a few additional sutures. All these sutures should be of linen, except that used to fix the catheter in the gastric opening; by using plain catgut for this, the suture will be absorbed in four or five days, when the catheter may be removed, washed, and replaced; since by this time the adhesions of the stomach to the anterior abdominal wall will make the temporary removal of the catheter quite safe. The catheter

should be clamped at its outer end to prevent leakage of gastric contents through it during the remaining steps of the operation. The stomach may now be sutured to the parietal peritoneum by three or four interrupted linen sutures, but as a rule it is unnecessary to attach the stomach to the abdominal wall, since by the time the tube is removed sufficient adhesions will have formed to prevent leakage into the unprotected peritoneal cavity. The abdominal wound is to be closed in the usual way without drainage, except for such drainage as takes place along the tract of the catheter.

(*b*) **Stamm's Method** (1894).[1]—A small incision, just large enough to admit the catheter, is made in the anterior gastric wall; the catheter (its outer end clamped) is inserted for about 2 cm. inside the cavity of the stomach, and is fixed in the gastric wall by a single suture of catgut (Fig. 89). Then a purse-string suture of linen is taken in the stomach wall, circularly around the catheter, and about 2 cm. distant from it; as this suture is tightened the catheter is pushed toward the cavity of the stomach, carrying with it the incision in the stomach wall, and thus inverting the gastric wall so that the catheter lies in a serous channel. One or two other purse-string sutures are similarly passed, and as each is tightened the inverted cone of gastric wall is lengthened, so that finally the catheter lies in a channel of more than 3 cm. in length (Fig. 89,C). The abdominal wound is then closed in layers around the catheter.

(*c*) **Kader's Method.**—The catheter is fixed in the gastric wall as in the previous operation, and the wall of the stomach is then inverted by a series of Lembert sutures of linen passed on opposite sides of the catheter; two sutures are passed above the catheter, each picking up the sero-muscular coats of the stomach in two places, so as to form two ridges with a groove between them; two other sutures are similarly placed below the tube; then, as this first series (consisting of four sutures) is tightened, the catheter is pushed inward, and, carrying the gastric wall with it, comes to lie in a serous channel as in the operations previously described. Two or three layers of these sutures are necessary to invert enough of the gastric wall, each newly applied series burying the preceding sutures. The abdominal wound is closed in the usual way.

Remarks.—Of these three operations, Stamm's method is the simplest, and requires less of the gastric wall for its successful per-

[1] E. J. Senn's operation (1896) consisted in drawing out a cone shaped portion of the anterior gastric wall, and maintaining it in this form by the application of purse-string sutures; the gastric fistula was thus lined by mucous membrane—just the reverse of Stamm's method.

formance than either of the others. This is an important point when the stomach is contracted from long disuse owing to esophageal or cardiac obstruction. We prefer it to all other methods, in ordinary

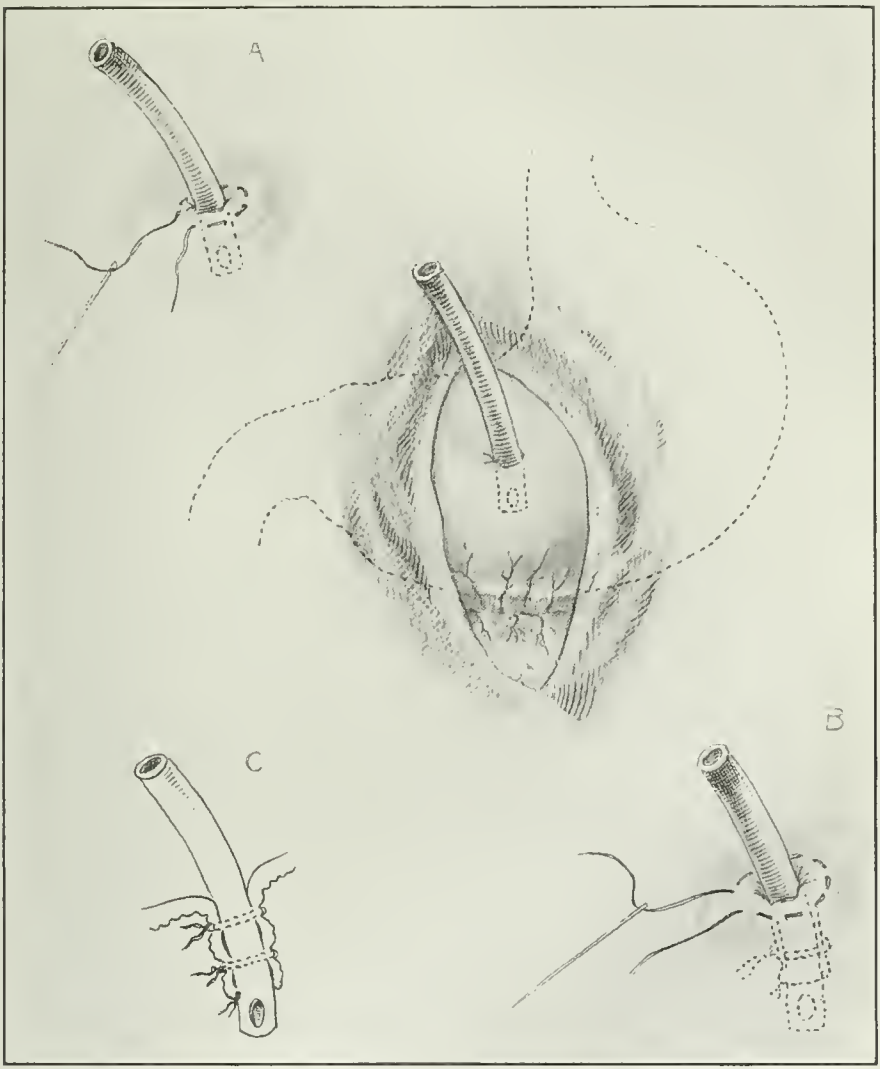


FIG. 89.—Stamm's Method of Gastrostomy: the Tube is Fixed to the Gastric Opening by a Single Suture of Plain Catgut. A Purse-string Suture of Linen is then Applied and Drawn Taut (Fig. A). A Second and Sometimes a Third Purse-string is Inserted (Fig. B). The Relation of the Tube to the Inverted Gastric Wall is Indicated in Fig. C.

cases. The channel formed from the cavity of the stomach to the skin in all these operations is usually absolutely continent so long as the catheter is in place; and unless the catheter remains in the fistula

for some months after the operation the channel is prone to become entirely obliterated from adhesion of its serous surfaces if the catheter is removed and not replaced for a week or ten days. Continence during the absence of the catheter from the fistula usually improves some months after the operation, and as the serous lining will be pretty well obliterated by this time, no fear of spontaneous closure need be entertained if the catheter is left out of the fistula between meal times.

2. Tavel's Operation (1906) has for its object the formation of a continent gastric fistula, lined by mucous membrane. An incision is made through the left rectus muscle, about four inches (10 cm.) in length, and a well-nourished loop of the upper jejunum is selected, provided with a long mesentery. The jejunum is then divided in two places, about 12 cm. apart, both sides of each section being guarded by rubber-covered clamps. The intervening portion of intestine is then excluded by doing an end-to-end anastomosis of the upper and lower segments. Either sutures or the Murphy button may be used for this purpose. The excluded segment is then transplanted through the transverse mesocolon into the lesser peritoneal cavity, and through the gastro-colic omentum out again into the upper portion of the general peritoneal cavity. The anal end of the excluded segment (which must be carefully distinguished from the upper or duodenal end) is then sutured into the anterior gastric wall (end to side implantation), and the duodenal end of the gut is sutured into the abdominal wound. The remaining portion of the abdominal incision is then closed in the usual manner. The peristaltic action of the bowel thus tends toward the stomach, and a continent fistula is established, lined with mucous membrane, and therefore having no tendency to contract.

Remarks.—Although we have had no personal experience with this operation, having no reason to be dissatisfied with the results obtained by Stamm's and by Witzel's methods, the few reported cases in which Tavel's method has been adopted have done well, and the fistula has entirely fulfilled the expectations of its inventor. It should, however, be remarked, that the operation is in itself a more serious undertaking than those already discussed, and that the time consumed, apart from the shock of an intestinal resection, will be a decided contraindication in the case of many patients in whom some form of gastrostomy must be done. Lambotte (1908) is said to have employed Tavel's operation successfully in two patients.

3. Operations of Esophagoplasty. (*a*) **Roux's Operation.**—The purpose of this operation is to create a new esophagus by transplanting a segment of the jejunum into the subcutaneous tissue over the ster-

num, and finally joining its upper end to the esophagus above the stricture, and its lower end to the stomach. It is interesting to trace the development of an operation seemingly so complicated. In 1894 Bircher attempted in two patients to create a channel, lined by skin, over the sternum, by means of a plastic operation on the skin, with the idea that this channel should serve as an artificial esophagus, by being joined above to the gullet, and below to the stomach. In 1904 Wullstein proposed an operation described as "ante-thoracic esophago-jejunostomy." He worked out the operation on the cadaver thus: he divided the jejunum, did an anastomosis in-Y, drew the distal loop of intestine through the transverse mesocolon and the gastro-colic omentum, and sutured it to the skin of the epigastrium. The cervical esophagus was to be connectd with this jejunal fistula by a rubber tube. Six months later Gluck operated upon a patient, joining an esophageal cervical fistula to a gastric fistula. Baudouin, ignorant of others' work, proposed a similar operation in 1907.

Roux (1907) operated in the following manner:

Selecting a portion of jejunum provided with a long mesentery, he divided the bowel in two places far enough apart to allow of the intervening portion reaching from the stomach to the patient's neck. He then re-established the intestinal canal by means of a Murphy button, and withdrew the excluded loop from the abdomen after detaching only the upper two-thirds or so of its mesentery. Owing to the anatomical distribution of the blood vessels in the upper jejunum this is quite easily accomplished. After implanting the distal end of the excluded jejunal loop into the anterior wall of the stomach, the patient was fed through the transplanted jejunum, before the subsequent steps of the operation were undertaken. A subcutaneous channel was next made from the upper angle of the abdominal wound at the ensiform process to the upper sternal region, and the loop of jejunum was carefully drawn up through this channel, and its upper end sutured to the skin. The arterioles in the gut thus transplanted continued to beat normally. A stomach tube was passed down through the bowel from the neck into the stomach, and allowed to remain in place several days, to facilitate feeding while the bowel acquired firm attachments in its new situation. The progress of the case was uneventful. The child was ready to be up when the case was reported. Only a little mucus was exuded from the fistula in the neck, and no gastric regurgitation was ever observed. The operation as planned was to be concluded at a second sitting, in which the esophagus above the stricture was to be united to the jejuno-gastric fistula.

This operation as devised by Roux had been carried out, according to Herzen (1908), by Kocher, Lambotte, and Gramse, as well as by Herzen himself. All these patients had carcinomatous obstruction of the cardia or esophagus, and all succumbed, though Herzen's patient lived until the fourth day. Lexer (1911) adopted the operation in a case of impermeable stricture of the esophagus, the jejunal tube having been united to the cervical esophagus by inverting a cylinder of skin over the upper end of the sternum; his patient was still using the new esophagus with satisfaction a year after operation and according to a recent note in the *Centralblatt für Chirurgie* (1920, xlviii, 509) is still living. Frangenheim (1911) also reported a successful operation of this nature.

(*b*) **Herzen,** of Moskow, modified (1908) this operation of Roux by transplanting the excluded jejunal loop through the transverse mesocolon and the gastro-colic omentum, as in Wullstein's operation described above; and by dividing the operation into three stages. The first step is to do "Jejunostomia retro-colica ante-thoracica cervicalis ypsiliformis," much as in Wullstein's operation. The second stage consists in dividing the transplanted segment of jejunum above the Y-anastomosis, closing the distal end, resecting any redundant portion of the proximal loop, and implanting the anal end of the proximal loop into the stomach. At the third operation, the cervical esophagus is united to the duodenal end of the jejunal loop, which was sutured to the skin of the cervical region at the first operation.

Herzen did the first stage of his modification of Roux's operation (retro-colic ante-thoracic cervical jejunostomy in-Y) on a very weak patient with cancer, who felt so much better after being fed through the jejunal fistula that he refused further treatment. The entire operation, in three stages, was done on another patient, on Sept. 10, Oct. 4, and Nov. 17, 1907 (the cervical esophagus and the transplanted jejunum being joined by end-to-end anastomosis), with entire success, a small esophageal fistula closing in three weeks, and the patient being in good health four weeks later, when he was shown to the Congress, and easily swallowed bread, meat-hash, eggs, etc. Intestinal peristalsis was visible under the skin of the thorax.

(*c*) **Jianu,** of Bucarest, in 1912, described a similar method of esophagoplasty, in which a tube was constructed from the greater curvature of the stomach itself, instead of from the jejunum. The stomach is drawn forward, and the gastrocolic omentum divided from near the pylorus well up to the fundus of the stomach, the line of section passing between the gastro-epiploic vessels and the colon.

Then the anterior and posterior walls of the stomach are incised parallel with the greater curvature, a long flap nourished by the left gastro-epiploic artery being raised, with attachment at the fundus (Fig. 90). The stomach opening and the free edges of the flap are closed by suture, and the tubular canal is brought out at the upper end of the abdominal incision, and carried up beneath the skin covering the sternum as far as it will reach. If care has been taken to moblize the fundus of the stomach sufficiently by division of the gastro-splenic and gastro-phrenic ligaments, the tube should reach almost to the neck. The advantage of cutting the end of the tube from the pyloric portion of

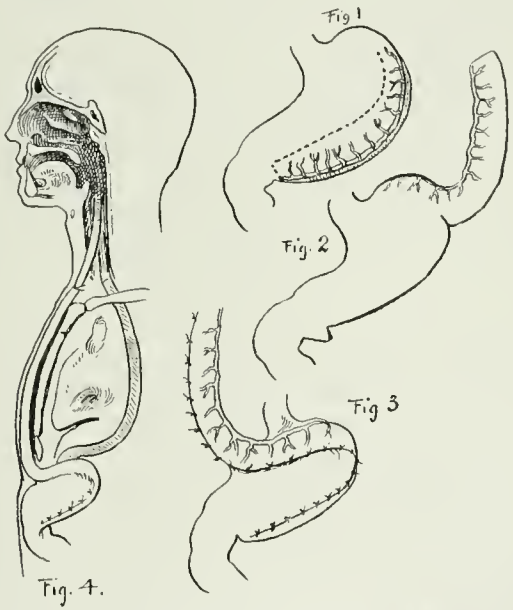


FIG. 90.—Diagrams Representing the Steps in Jianu's Method of Esophagoplasty. Fig. 1 Indicates the Incision in the Stomach Parallel to Its Greater Curvature. Fig. 2 Shows the New Esophagus Constructed from the Greater Curvature. Fig. 3 Shows the New Esophagus Rotated Toward the Midline. Fig. 4 Shows It Placed Subcutaneously over the Sternum and United with the Cervical Esophagus.

the stomach is that the secretions of its mucous membrane, not being acid, will not digest the skin of the neck around the opening. Subsequently an anastomosis is made with the cervical esophagus either directly, or indirectly by constructing a plastic skin channel.

In 1914 Jianu described two cases in which he had employed this operation successfully in children. W. Meyer (1914) has used the operation in three cases with good results. He thinks it important to make the orifice between tube and cavity of the stomach small, and to cauterize the branches of the pneumogastric nerves which supply

the tube, so as to prevent regurgitation from the stomach. Jianu says if regurgitation occurs it is due to faulty technique—not constructing the base of the tube high enough in the fundus of the stomach.[1]

Remarks.—That the methods of Herzen and Jianu are improvements over that of Roux is probably true; but the same objection lies against both that we raised against Tavel's method, namely, that in most patients such a severe operation will kill. If the operation is to be undertaken for the relief of carcinomatous stenoses this fact may not be thought to be an objection; but the surgeon is not an executioner. It does seem, however, that such an operation as this may well be of use in cases of impermeable benign stricture of the esophagus, which have hitherto baffled surgeons completely. Internal esophagotomy and retrograde dilatation in some patients will continue to fail in the future as they have in the past to relieve the deplorable condition of those who must feed themselves through a gastric fistula; and in such patients, whose general health is good, but in whom no other means of cure is available, the operations of Roux, Herzen and Jianu may be perfectly justifiable.

Pyloroplasty

Inasmuch as the inefficiency of the old-fashioned **pyloroplasty** has been abundantly demonstrated, it is our intention to describe only Finney's modification. Although Finney still prefers to use the sutures as shown in the accompanying figures, most surgeons today employ rubber covered clamps, as in other operations upon the stomach and intestines. The main points on which stress is laid by Finney, are the very thorough separation of the peri-pyloric adhesions, and the large size of the gastro-duodenal incision, which should be not less than 12 cm. (over four inches and a half).

The operation is described by Finney as follows (Trans. Amer. Surg. Assoc., 1902, xx, 165): "Divide the adhesions binding the pylorus to the neighboring structures, also free as thoroughly as possible the pyloric end of the stomach and first portion of the duodenum. Upon the thoroughness with which the pylorus, lower end of the stomach and upper end of the duodenum are freed, depends in

[1] According to a recent note in the Centr. f. Chir. (1920, xlviii, 509) a number of similar operations have been done within recent years in Germany: Axhausen, 5 cases, 1 death; Hinz, 2 cases, 1 death; Hirschmann, 2 cases, results not given. Kirschner is quoted as having freed the stomach from *all its connections* except with the duodenum, *preserving only the latter, and the gastric and the right epiploic arteries;* the stomach is then drawn up extra-thoracically to the cervical esophagus, with which the fundus of the stomach (not the cardia) is directly united. His patient recovered.

large measure the success of the operation and the ease and rapidity

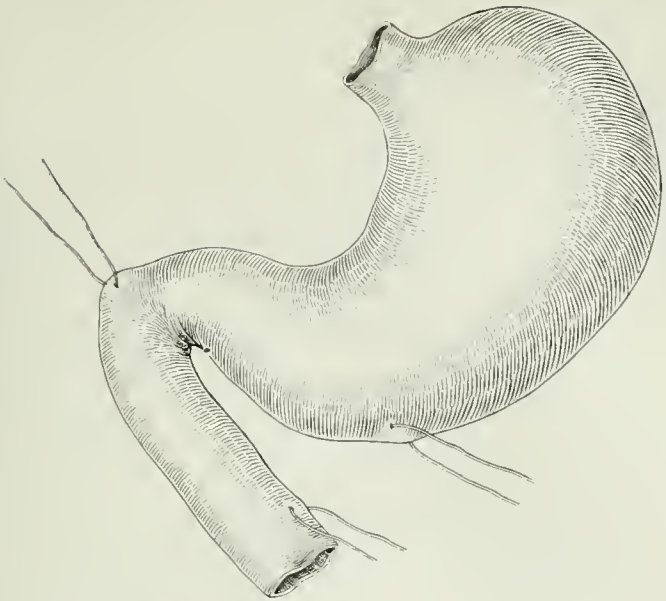


FIG. 91.—Finney's Method of Pyloroplasty. The Traction Sutures Inserted.

of its performance. I wish to emphasize this as one of the most important points in the operation. Frequently at first sight the pylorus

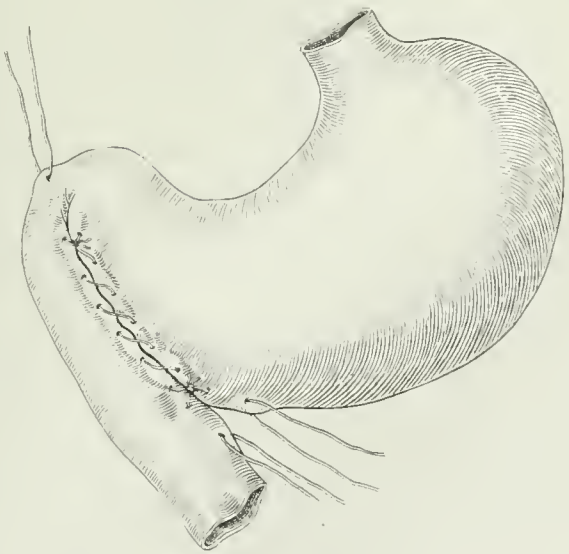


FIG. 92.—Finney's Pyloroplasty. The Posterior Continuous Sero-serous Suture is Applied.

may seem hopelessly bound down, when, after a little patient toil and

the judicious use of the scalpel and blunt dissector, it is found that it can be freed with comparative ease. A suture, to be used as a retractor, is taken in the upper wall of the pylorus, which is then retracted upward. A second suture is then inserted into the anterior wall of the stomach, and a third into the anterior wall of the duodenum at equidistant points, say about 12 cm. from the suture just described in the pylorus. These second sutures mark the lower end of the gastric and duodenal incisions, respectively. They should be placed as low as possible in order that the new pylorus may be amply large. Traction is then made upward on the pyloric suture, and downward

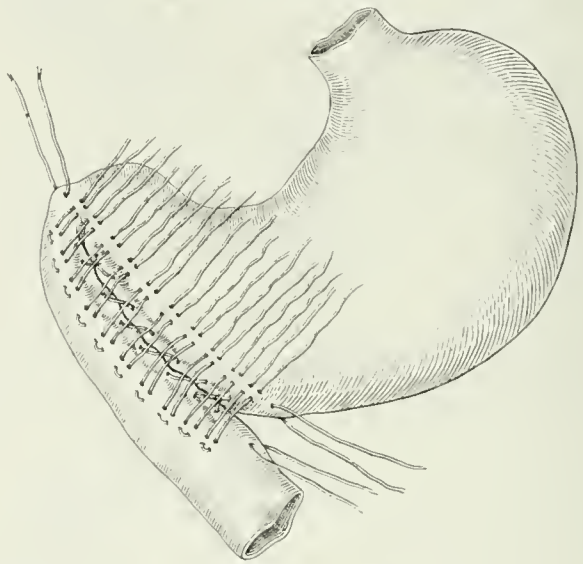


FIG. 93.— Finney's Pyloroplasty. The Mattress Sutures Inserted.

in the same plane, on the gastric and duodenal sutures. This keeps the stomach and the duodenal wall taut, and allows the placing of the sutures with greater facility than if the walls remained lax (Fig. 91). The peritoneal surfaces of the duodenum and stomach along its greater curvature are then sutured together as far posteriorly as possible (Fig. 92). For this row I would recommend the use of the continuous suture, as it is more easily and quickly applied, and it can be reinforced after the stomach and duodenum have been incised. After the posterior line of sutures has been placed, an anterior row of mattress sutures is taken, which are not tied, but left long, in the manner indicated in Fig. 93. These sutures, after they have been placed, are retracted vertically in either direction from the middle of the portion included in the row of sutures (Fig. 94). Then, after all the stitches have been placed and retracted, the incision is made in

the shape of a horseshoe. The sutures should be placed far enough apart to give ample room for the incision. The gastric arm of the incision is made through the stomach wall just inside the lowest point of the line of sutures, and is carried up to and through the pylorus and around into the duodenum, down to the corresponding point on the duodenal side. Hemorrhage is then stopped. It is well to excise as much as possible of the scar tissue upon either side of the incision in order to limit as far as possible the subsequent contraction of the cicatrix. It is well, too, to trim off with scissors redundant edges of

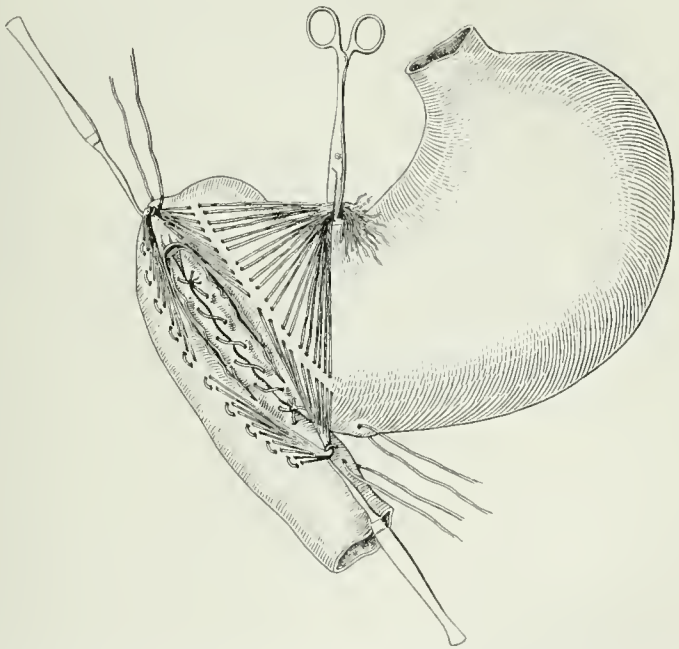


FIG. 94.—Finney's Pyloroplasty. The Mattress Sutures Having Been Drawn Upward and Downward, the Stomach and Duodenum are Opened by a Long Hair Pin Shaped Incision.

mucous membrane at the new pylorus. A continuous catgut suture is now taken through-and-through all the coats of the intestine on the posterior side of the incision (Fig. 95). This reinforces the posterior line of sutures, secures better approximation of the cut edges of the mucous membrane, and prevents the reunion of the divided intestinal walls. The anterior sutures are then straightened and tied, and the operation is complete, unless one wishes to reinforce the mattress sutures with a few Lembert stitches" (Fig. 96).

The only modifications which we have adopted in the limited

number of cases in which this operation has been done, consist in (1) the use of clamps; (2) continuing the through-and-through catgut sutures all around the gastro-intestinal anastomosis, as in other forms of lateral anastomosis, instead of only on the posterior surfaces of the incision, as recommended by Finney; and (3) in omitting the anterior row of mattress sutures, their place being taken by a continuation of the posterior row of Lembert sutures first applied. In other words, we do the ordinary operation of lateral anastomosis, as in gastro-enterostomy or entero-enterostomy.

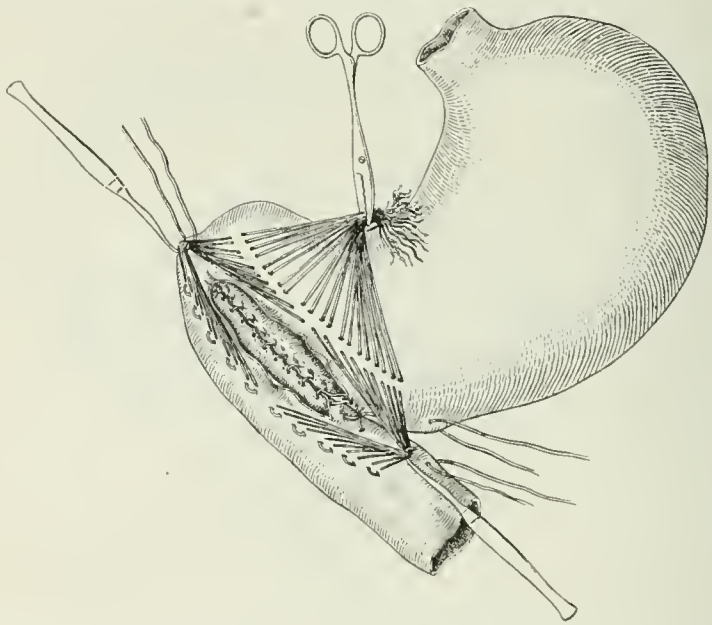


FIG. 95.—Finney's Pyloroplasty. The Adjacent Margins of the Gastric and Duodenal Incisions Have Been United by a Continuous Through-and-through Suture.

Durante's Pyloroplasty.—According to Ricard and Chevrier (1905) Durante adopted a form of pyloroplasty in which a Y-shaped incision is made through the pyloric valve and the pyloric portion of the stomach. The stem of the Y divides the pylorus, and into the incision thus made, the triangular flap included between the branches of the Y is drawn and sutured, thus increasing the diameter of the pylorus at the expense of the anterior gastric wall. The principle is the same as in Nicoll's operation for infantile stenosis of the pylorus (p. 137).

Kocher's Method of Lateral Gastro-duodenostomy is rendered possible by mobilization of the duodenum, adopted years ago by

Finney, and subsequently popularized by Kocher. Leriche (1906) gives the history of this preliminary step, the idea of mobilizing the duodenum apparently having originated with Terrier. The operation resembles that of Finney, except that the pylorus itself is not divided.

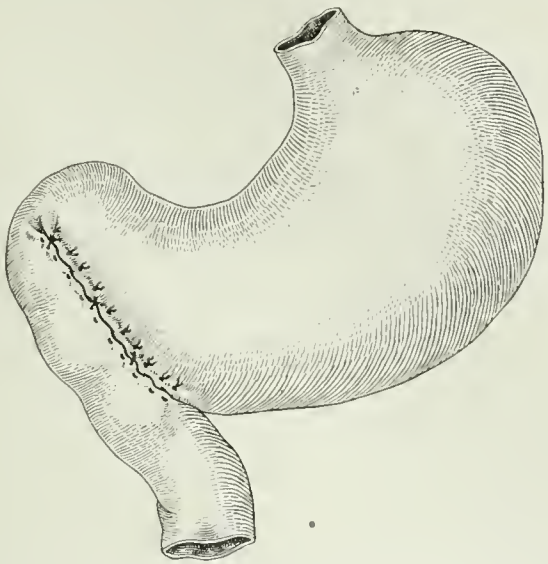


FIG. 96.—Finney's Pyloroplasty: the Operation is Completed by Straightening Out and Tying the Mattress Sutures.

GASTRO-JEJUNOSTOMY

Historical.—The operation was first performed in 1881 (at the suggestion of his assistant Nicoladoni), by Wölfler. The case was one of pyloric carcinoma, and when this was found to be inoperable, Wölfler was about to close the abdomen, when Nicoladoni suggested that by anastomosing the small bowel with the anterior wall of the stomach a new exit for the food would be provided. Although in this original operation the jejunum was attached to the stomach in an anti-peristaltic direction (that is, with its anal end toward the cardiac end of the stomach), yet it is customary to speak in general of all anterior gastro-jejunostomies as "Wölfler's method." In 1887 Rockwitz introduced an operation in which, by attaching the jejunum to the stomach in the other direction, "iso-peristaltic" action was obtained. In 1885 v. Hacker published a method of gastro-jejunostomy by which the anastomosis was made in the posterior wall of the stomach, through an opening in the transverse mesocolon. He used an afferent loop of jejunum about 35 cm. in length. Since that time all posterior

trans-mesocolic gastro-jejunostomies by lateral anastomosis have been described in general as by v. Hacker's method.

The idea of a gastro-jejunostomy in-Y is attributed by Roux (1897) to Socin; but the latter credits its origin to Wölfler, who in his "second" method adopted this technique in connection with anterior gastro-jejunostomy. Roux (1897) calls his own method "posterior retro-colic gastro-enterostomy in-Y." (Fig. 105.)

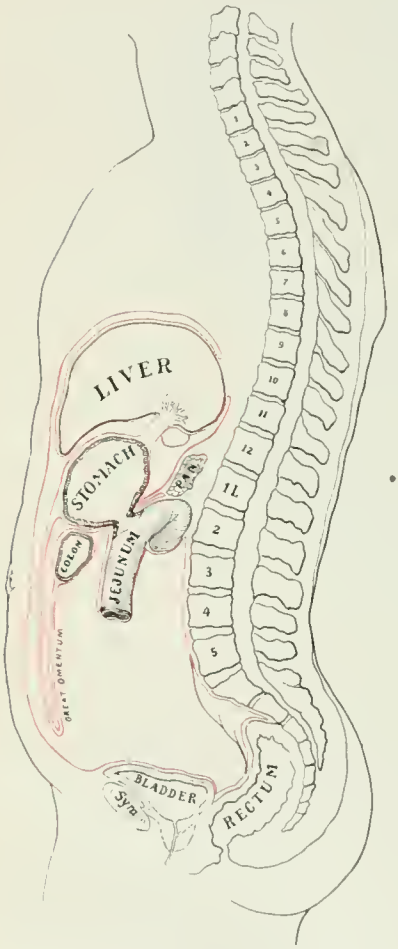


FIG. 97.—Posterior "No-loop" Gastro-jejunostomy.

The idea of doing an entero-anastomosis between the afferent and efferent loops of the jejunum, supposed to prevent the discharge of the duodenal secretions into the stomach, is due to Lauenstein, who suggested in 1891 the anastomosis of the afferent loop with a neighboring coil of intestine. Braun in 1892 adopted as his method an anastomosis between the afferent and efferent loops; while Jaboulay, in the same year, anastomosed the jejunum below the gastro-jejunal anastomosis with the third portion of the duodenum. To make certain that the contents of the afferent loop would pass directly into the efferent, through the entero-anastomosis, and not continue past it into the stomach, the afferent loop (between the stomach and the entero-anastomosis) was divided and both ends closed by Doyen in 1898; while Fowler, in 1902, was content to ligate the afferent loop with silver wire. Lücke in 1899 advocated as an improvement on Doyen's method an operation (Lücke's second method) which combined the merits of the Y-operation with the advantages of the enteroanastomosis introduced by Braun. To accomplish this, Lücke divided the jejunum completely 25 to 40 cm. (10 to 15 inches) from its origin, closed both ends, and then made two lateral anastomoses—one between the posterior gastric wall

and the distal segment of jejunum, and the other between the two segments of the jejunum, a convenient distance below the stomach. By this means he avoided the end to side implantations of Roux's method, which he regarded as dangerous, while at the same time he retained the advantage of the principle of the Y-anastomosis, which rendered a simultaneous entero-anastomosis unnecessary.

The most valuable modification of all was that introduced in 1901 by Petersen, of Czerny's clinic: in this operation, a posterior trans-mesocolic gastro-jejunostomy, the afferent loop was abolished, the anastomosis in the jejunum being made as close as possible to the duodeno-jejunal juncture and the bowel was affixed to the stomach in the long axis of the patient's body, that is, nearly at right angles to the greater curvature (Figs. 97, 98). This remains the standard operation, and has superseded the "short loop method"[1] formerly taught by Robson, Moynihan, Scudder, and Mayo.

It is our purpose to describe only the following methods of performing gastro-jejunostomy. 1. Posterior trans-mesocolic gastro-jejunostomy without a loop. 2. Anterior ante-colic gastro-jejunostomy with clamps or the Murphy button. 3. Roux's gastro-jejunostomy in-Y.

The indications for the employment of gastro-jejunostomy have been discussed in connection with the various affections for which it may be adopted.

General Considerations.—The same *incision* will suffice no matter what method be adopted. That most frequently employed is a longitudinal incision through the right rectus muscle, close to the median line. It should be about four inches (10 cm.) in length, extending from below the ensiform process nearly to the umbilicus. A very careful examination should be made of the whole operative field before commencing the gastro-intestinal anastomosis, since it occasionally happens that some other method than that originally designed will be required to meet the condition found. Especially important is it to determine the extent of the whole stomach, for, as Moynihan has pointed out, neglect of this precaution may result in the surgeon overlooking the existence of an hour-glass stomach with small cardiac pouch. We think the preference of the operator should always be for a posterior gastro-jejunostomy; hence his next step should be to determine whether the posterior wall of the stomach is accessible through the transverse mesocolon. Before proceeding with this search, the entire skin sur-

[1] "Short-loop method" to distinguish it from the original "long-loop method" of v. Hacker.

face surrounding the abdominal incision should be covered with hot moist gauze pads, in order to protect any viscera which may have to be drawn out of the abdomen. By now drawing the great omentum with its attached transverse colon into the wound, and turning them upward on to the hot gauze pads already placed over the epigastrium and lower thorax, the transverse mesocolon is readily brought to view. If the stomach is densely adherent to the mesocolon and the pancreas it will be impossible to withdraw the transverse colon in this way; but even after this manœuvre has been easily accomplished and the posterior gastric wall exposed by division of the mesocolon, it may be found that there is not a sufficient area of healthy gastric wall to permit of an anastomosis being made in its posterior surface. Under these circumstances the upper coil of jejunum should be identified before replacing the transverse colon; neglect of this precaution may result in the surgeon subsequently selecting the wrong coil of small intestine for his anastomosis. We know of several instances in which accomplished operators have by mistake anastomosed the lower ileum to the stomach. By pulling the transverse colon and the attached great omentum, as already described, out of the abdominal wound, the primary coil of the jejunum is readily brought into view (Fig. 98).

It is important, whenever possible, to make the anastomosis near the pyloric portion of the stomach, so that the new opening shall resemble the pylorus as nearly as possible in its physiological action.

Before the gastro-intestinal anastomosis is commenced, the surgeon should isolate by the use of gauze packs the immediate structures involved, and all viscera outside of the abdomen must be carefully covered with hot moist gauze. One piece of gauze should always be passed just beneath the site of the proposed anastomosis, to be withdrawn on its completion. Usually, after the posterior wall of the stomach has been exposed through the transverse mesocolon, it will be found possible to replace within the abdomen both the transverse colon and the stomach itself, before proceeding with the operation.

Before beginning any operation in which it is proposed to use the Murphy button, its mechanism should be scrupulously and repeatedly tested by the *surgeon himself*. The lumen of each half of the button should be filled with cacao butter; this prevents escape of visceral contents, but will be melted by the heat of the body a few moments after the anastomosis has been completed (Hartmann).

Posterior Gastro-jejunostomy.—The transverse colon and the attached omentum are drawn out of the wound, and by pulling these structures upward and to the patient's right the transverse mesocolon

is put upon the stretch and the origin of the jejunum brought into sight. This is then caught lightly in toothed forceps, placed on its antimesenteric border, about 5 cm. apart, and the right portion of the three-bladed intestinal anastomosis forceps is adjusted so as to include at least 10 cm. of the jejunum in its grasp (Fig. 98).

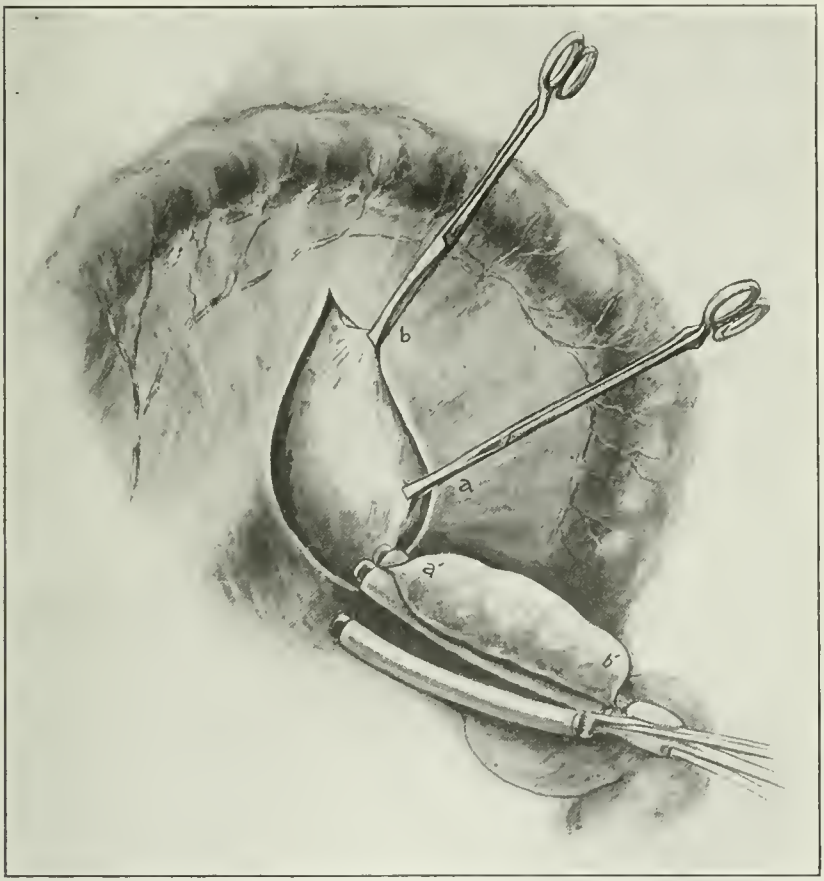


FIG. 98.—Posterior Transmesocolic Gastro-jejunostomy. The Transverse Colon has been Drawn from the Wound, Making Taut the Transverse Mesocolon, which has Been Opened, Exposing the Posterior Wall of the Stomach; the Jejunum is to be Anastomosed to the Stomach (a to a′ and b to b′).

The point of emergence of the jejunum from the transverse mesocolon corresponds in most instances very closely to the duodeno-jejunal juncture; but in a small proportion of cases the jejunum is retro-peritoneal for a variable distance from its origin before leaving the posterior parietal peritoneum and becoming invested by the mesentery. It is important for the surgeon, therefore, critically to

examine the supposed primary coil of jejunum, and to make sure that no abnormality will cause him to perform a long loop operation when he aims to leave no loop at all. If a peritoneal fold binding the jejunum to the mesocolon is recognized, it should be divided up to its origin and the anastomosis may be made in the raw area left on the jejunum.

Selecting now a bloodless area to the left of the main trunk of the middle colic artery, the transverse meso-colon is picked up with dissecting forceps and carefully divided, and the opening enlarged in the sagittal plane, by a few snips with scissors, until it is about 8 or 10 cm. in length. The left hand of the operator, which holds the transverse colon between finger and thumb, the fingers being on the upper surface, can now make the posterior gastric wall protrude into this opening in the transverse mesocolon. It will usually be found that the portion of the gastric wall thus brought to view is that immediately beneath the lesser curvature and that it is quite close to the greater curvature of the stomach. The stomach wall is caught by toothed forceps, or by light ring-bladed rubber covered forceps, in two places, about 5 cm. apart, picking up a fold of the stomach which runs in the long axis of the patient's body (Fig. 98). The base of the fold of gastric wall thus grasped should be from 8 to 10 cm. in length and the portion grasped should be in the pyloric portion of the stomach. The clamp should not be applied parallel to the greater curvature of the stomach, since to do so would markedly distort the jejunum, which at its origin runs more or less perpendicularly to the greater curvature of the stomach (Fig. 103). Mayo urged (1906) that the incision be made obliquely downward and to the left, claiming that this preserves the normal anatomical relations better, and that the fact of the anti-peristaltic direction of the anastomosis thus effected is of no consequence. But as Moynihan (1908) pointed out, the jejunum is quite freely movable below the ligament of Treitz, and if found running downward and to the left while patients are on their backs, may also be found running downward and to the right if they are made to lie on their right side. Therefore it seems best to adhere to the method of Petersen, who made the opening in the stomach practically at right angles to its greater curvature, or perhaps inclined a little to the right Moynihan laid stress on the importance of not rotating the jejunum on its long axis, as this may cause obstruction at the duodeno-jejunal flexure. A small piece of gauze is then laid beneath the parts to be approximated, and the left blades of the three-bladed clamps are adjusted to the stomach in such a manner that the aboral end of jejunum corresponds with the greater curvature of the stomach, and its duodenal end with the lesser curvature of

the stomach (Fig. 98, a with a′, b with b′). The transverse colon and all the viscera not immediately concerned in the anastomosis are then replaced inside the abdomen, and the entire operative field is isolated by sterile gauze. The stomach and the jejunum, which are maintained in apposition by the clamps, are now to be united by a posterior sero-serous continuous suture of linen thread. This suture should be applied as close as possible to the blades of the clamp, so as to leave plenty of room for the through-and through sutures. The posterior row of sutures should commence a little beyond one extremity of the proposed anastomosis, and is to be continued a little past the other end, where it may be knotted to prevent puckering of the anastomosis (as advised

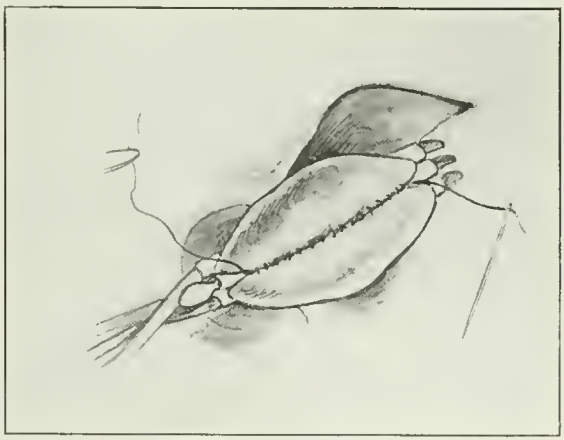


Fig. 99.

by Hartmann). The ends of this suture should be left long, and, with the needle still threaded, it should be laid aside and covered with sterile gauze until again needed (Fig. 99).

An incision about 6 cm. long is now made with a scalpel through the serous and muscular coats of the stomach, about one centimetre distant from the continuous suture just inserted. When the mucous layer of the gastric wall is thus exposed, it will pout a little into the incision; the jejunum should be opened in a similar manner, and for an equal distance. Before either stomach or jejunum is opened it is the custom of the senior author at present to suture together the adjoining lips of the incisions in the stomach and jejunum, with a continuous suture of chromic gut. In this he follows the teaching of Mayo, believing that better hemostasis is thus secured. It may be that this

step will be abandoned in the future as unnecessary.[1] Then the mucous coats of stomach and jejunum are opened, by picking them up in forceps and cautiously cutting into their lumens with scalpel. The incisions in the mucosa are then enlarged with scissors the full length. If redundant some of the pouting mucous membrane may be excised. It is always more redundant in the jejunum than in the stomach.

The surgeon now has the cavity of the stomach and that of the jejunum opened. He should next unite the adjacent free edges of these viscera by a through-and-through continuous suture of chromicised (or iodized) catgut. Beginning at one extremity of the incisions into stomach and jejunum, the needle is passed from the mucous

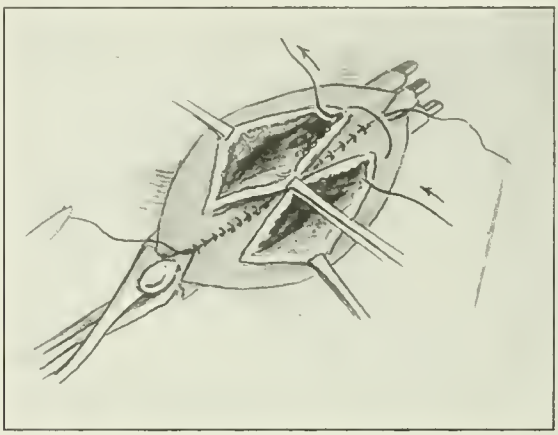


Fig. 100.

surface of the jejunum through its wall to its serous surface, and from the serous surface of the stomach into the cavity of the latter. The suture is then fixed by knotting it; and by continuing to suture in a precisely similar manner the posterior margins of the incisions are united from one extremity to the other (Figs. 100, 101). The suture may be knotted from time to time if desired, to prevent puckering, as taught by Hartmann; usually this is unnecessary. When the surgeon reaches the end of the posterior margins of the gastric and jejunal incisions, he should continue his suture carefully around the end (Fig. 102, *A*), and begin the approximation of their anterior margins. As these are not held in close

[1] Certainly it should be unnecessary, theoretically, as a continuous overhand through-and-through suture such as is currently employed here by most operators is more of a safeguard against bleeding from the cut edges of stomach and bowel, than is the right-angled through-and-through suture which is employed to unite the anterior lips of the anastomosis where the danger from bleeding is equal, and yet where Mayo considers no extra hemostatic suture is necessary.

apposition by the clamps, as were the posterior margins, it is a little difficult at first to understand how to continue the suture so as to secure the approximation of serosa to serosa. If the surgeon, however, pursues precisely the same method already adopted, he will have no trouble with this part of the operation; he should pass his needle from the mucous surface of the jejunum to its serous surface, then from the serous surface of the stomach to its mucous surface, draw the stitch tight, and repeat the process. To put it briefly, the needle is passed *out*, *in*, and *over*; *out*, *in and over;* etc.—that is to say, *out* of one organ, *into* the other, and *over* the line of sutures, again to pass *out* of the jejunum, *into* the stomach, and across the suture line back to the

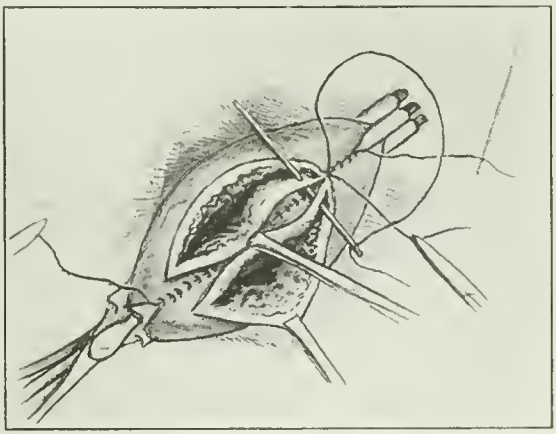


FIG. 101.

starting point. By drawing each stitch fairly tight, it is easy to determine the point where the needle should next be inserted. About four or five stitches should be made to every inch (or one to every 0.5 cm.) When the angle of the incision has been successfully turned in this manner, it is easier to secure inversion of the anterior lips of the anastomosis by employing a continuous right-angle suture of the type represented in Fig. 102, *B*: in this the needle after entering one organ from its serous to its mucous surface at once retraces its step, *leaving the loop of the suture on the mucous surface;* the needle is then carried across to the opposite lip of the anastomosis, entering it from its serous surface, at once retracing its course from mucous to serous surface, again *leaving the loop of the suture on the mucous surface.* This brings broad areas of serous surface into contact and prevents prolapse of the mucous membrane between the stitches. When the entire circumference of the anastomosis has been united by this through-and-through suture,

the catgut thread is tied to its own original end, at the starting point, and cut short. The sero-serous suture, previously laid aside, is now to be resumed, reinforcing the anastomosis on its anterior aspect, and

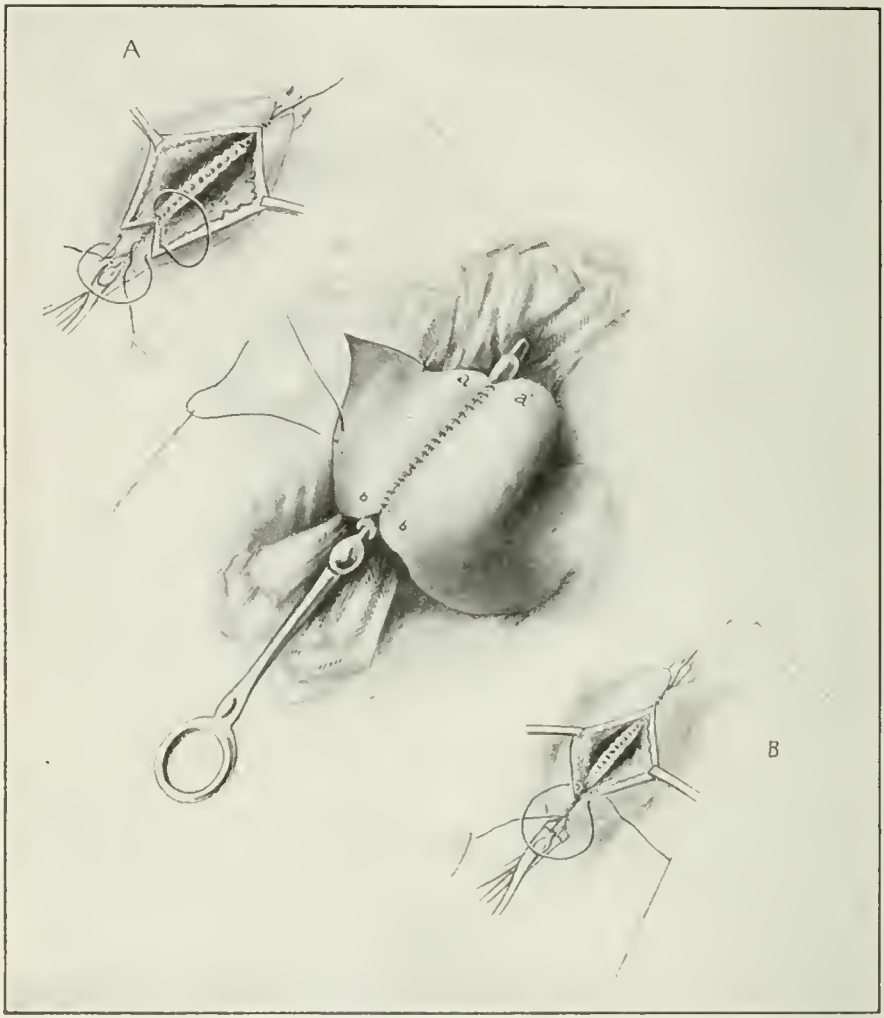


FIG. 102.—Posterior Gastro-jejunostomy. At A: the Through-and-through Suture is Shown Turning the Angle of the Incisions in Stomach and Jejunum; at B, It is Inverting the Anterior Lips, as a Through-and-through Right-angled Suture. The Central Drawing Shows the Anastomosis Completed by Continuing the Original Sero-serous Suture Back again to Its Starting Point. It also Indicates how the Cut Edges of the Mesocolon are Sutured to the Stomach.

completing the circumference of the wound to its starting point, where it is to be tied to its own initial extremity and cut short.

It is advisable, *as soon as the through-and-through sutures have been completed,* and all danger of contamination from gastric or intestinal

contents has been thus eliminated, *to release the rubber covered clamps;* for it is not desirable to keep them in place too long, as there is always a possibility of long continued pressure injuring the gastric or intestinal walls. It is convenient, however, to keep one blade beneath the anastomosis, as a sort of bridge to hold the viscera in place, until the anterior sero-serous sutures have been completed (Fig. 102). If any bleeding point is observed when the clamps are removed, it should be caught up in a special suture.

When the gastro-intestinal anastomosis has been finished in this manner, the surgeon should thoroughly rinse his gloved hands in corrosive sublimate and then in sterile water. It is often better to put on another pair of sterile gloves.

The edges of the opening in the mesocolon are next to be sutured to the gastric wall a short distance away from the anastomosis (Fig. 102). This is a very important step in the operation, and should never be forgotten. It prevents prolapse of coils of small bowel into the lesser peritoneal cavity, and also keeps the mesocolon from constricting the anastomosis itself or from slipping down over the afferent and efferent loops of jejunum and thus causing obstruction. It is well to place a stitch at each extremity of the opening in the mesocolon to keep the incision from tearing larger.

The gauze surrounding the field of operation is now to be removed; as that piece of gauze immediately underlying the anastomosis is drawn out it will rotate the anastomosed structures far enough to enable the surgeon to inspect the posterior line of sutures, and thus to assure himself that all is in good condition on that surface of the anastomosis.

The viscera are then carefully replaced in the abdomen, the great omentum is drawn down over the small intestines, and the abdominal wound is closed.

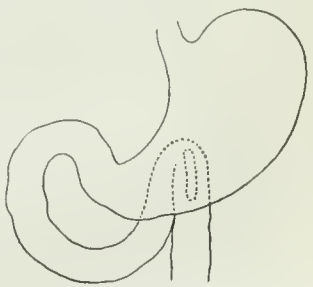


FIG. 103.—Diagram of Posterior No-loop Gastro-jejunostomy.

Anterior Gastro-jejunostomy.—The primary loop of the jejunum is identified by withdrawing the transverse colon from the wound, and seeking the duodeno-jejunal juncture in the transverse mesocolon just to the left of the spinal column. The transverse colon is then replaced in the abdomen, and a point on the jejunum is selected which will reach the anterior wall of the stomach without constricting the transverse colon. This is usually 40 to 60 cm. (16 to 24 inches) below the origin of the jejunum. It is not necessary, nor is it desirable, to

split the great omentum up to the transverse colon so as to render the use of a shorter loop possible but the jejunum should be brought up to the left of the omentum, which is displaced toward the patient's right (not as shown in Fig. 104 toward the patient's left). We prefer to make the anastomosis with the clamps, the technique employed being the same as that described under the heading of posterior gastro-jejunostomy (p. 338). If the **Murphy button** is to be used, a purse-string

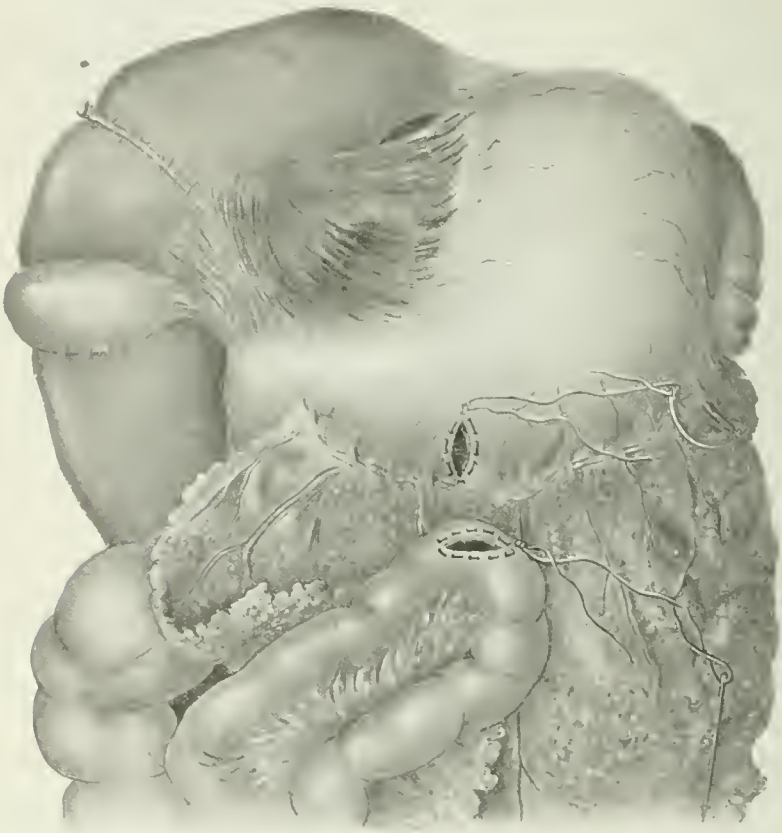


FIG. 104.—Anterior Gastro-jejunostomy with the Murphy Button.

suture of linen is inserted in a healthy portion of the anterior gastric wall, near the greater curvature, and if possible in the pyloric portion of the stomach. A similar purse-string suture is also applied to the jejunum, at the point selected, opposite the mesenteric attachment. Each of these sutures should encircle a space just large enough to permit of the introduction of a Murphy button; the sutures should not be tied.

and the ends should be left long, to permit of pulling the suture tight after each half of the button has been introduced.

After these sutures have both been placed, an incision should be made in the gastric wall within the circle formed by the purse-string suture, and the male half of the button, held in the bite of a hemostat, should be quickly passed into the incision. The gastric suture is then drawn tight, tied, and cut close. The jejunum is opened in a similar manner, the female half of the button is passed into the incision, is fixed by tying the pursestring suture; and then the two halves are approximated and pushed home. A few interrupted Lembert sutures, or a continuous suture, may then be introduced around the margins of the button. The packs are then withdrawn, the viscera suitably replaced in the abdomen, and the abdominal wound closed.

It is proper to note in this place that the Murphy button was not approved in recent years by its inventor for use in anterior gastro-jejunostomy. While it is true that the operation may be done equally well by the use of clamps and suture, without a button, yet when it is desired to complete the operation rapidly, we believe no method is so satisfactory as the use of the Murphy button. And although we employ the clamps whenever possible, we have elected to describe the use of the Murphy button in connection with anterior gastro-jejunostomy, because that is about the only form of gastro-intestinal anastomosis in which the surgeon cannot invariably dispense with such an aid. Murphy modified the button so that an oval instead of a circular opening may be made; but we have had no personal experience with this newer form.

Posterior Gastro-jejunostomy in-Y (Roux) (Fig. 105).—Deliver the transverse colon, and draw out of the abdomen the first coil of the jejunum, and empty it of its contents by manipulation with the fingers for a distance of about 30 cm. Apply a long rubber covered clamp across its lumen in two places, leaving an omega loop of at least 25 cm., with its mesentery, hanging free beyond the clamp. Or if it seems undesirable to clamp the entire blood supply for so long a time, the surgeon may employ two smaller clamps, each constricting the entire lumen of the jejunum, about 25 cm. distant one from the other. The proximal point of the jejunum clamped should be about 25 cm. below the duodeno-jejunal juncture. Next divide the jejunum completely across about 10 cm. below where the clamp compresses its proximal end. This will leave two segments of jejunum within the grasp of the clamp: the proximal segment will be about 10 cm. and the distal about 15

cm. long. The posterior wall of the stomach is then exposed by opening the transverse mesocolon, is caught in another pair of rubber covered anastomosis clamps, and is opened for a distance of 3 to 4 cm. Then the surgeon unites by terminolateral implantation the open circular end of the distal jejunal loop with the stomach, applying first a posterior sero-serous suture, then a through-and-through hemostatic suture, which passes entirely around the anastomosis; and finally the posterior sero-serous suture is resumed, re-inforcing the gastro-intestinal anastomosis on its anterior aspect. Then an incision is made in the distal segment of jejunum opposite the mesenteric attachment, about 2.5 cm. from the point where it is clamped, and the proximal segment is implanted into this incision, in precisely the same manner (terminolateral anastomosis) as that in which the jejunum has just been united to the stomach. The clamps may now be removed. If the gastric clamp be removed before the second anastomosis is completed there will be danger of leakage of gastric contents through the lateral incision made in the jejunum for the jejuno-jejunal anastomosis.

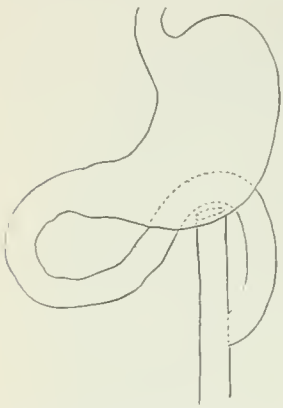


FIG. 105.—Diagram of Roux's Method of Posterior Gastro-jejunostomy in-Y.

Before closing the wound, the structures should be carefully arranged in the abdomen; and the surgeon should not neglect to suture the transverse mesocolon to the stomach around the gastro-jejunal anastomosis, nor to close any opening in the mesentery of the jejunum.

Exclusion of the pylorus was proposed and employed in 1895 by v. Eiselsberg. The operation consists in dividing the stomach completely in the pre-pyloric region, closing both ends by suture, and then performing posterior gastro-jejunostomy. Jonnesco (1907) preferred this form of operation to a simple gastro-enterostomy, and always employed it when excision (pylorectomy; partial gastrectomy) was impossible. But it has not met with general acceptance.

We have already expressed our preference for simple gastro-jejunostomy and we cannot see that this operation of exclusion of the pylorus presents any particular advantages in ordinary cases. When the pylorus is freely patulous, there will be a tendency for the gastric ulcers to heal when the acidity of the gastric secretions shall have been diminished by gastro-jejunostomy; but exclusion of the ulcerated area from the stomach would prevent this change in the gastric se-

cretions having any effect on the ulcers, while at the same time it would subject the patients to very nearly as much danger as would a pylorectomy.

Gastroplasty

Gastroplasty, an operation analogous to pyloroplasty, is adopted in certain cases of hourglass constriction of the stomach (Fig. 106). This operation is said to have been employed first by Bardeleben, in 1889. The first patient who recovered was operated upon in 1892 by Krukenberg.

A rubber covered clamp is applied to the stomach, with its axis corresponding to the long axis of this organ, so as to pick up in its grasp a fold of gastric wall forming the channel of communication between the two pouches of the stomach. This fold of gastric wall is then incised down to the mucous coat, which is excised when it pouts into the incision. Forceps are then used to grasp the margins of the gastric incision at its extremities and at the mid-point of each of its sides. As these forceps draw the gastric incision well upward, the clamp is loosened, removed, and reapplied at right angles to its former position. As this is done the pairs of forceps formerly at the mid-points of the gastric incision are separated so as to change the formerly longitudinal incision into a transverse wound, while the forceps formerly at the ends of the incision will now be attached to its sides. The rubber covered clamp having been reapplied, the gastric incision is closed by a through-and-through hemostatic suture of iodized catgut, which is afterward reinforced by a continuous Lembert suture of linen. The rubber covered clamps should be loosened as soon as the through-and-through suture has been completed, to test its hemostatic effect; and, as in other operations, any bleeding points should be controlled by separate sutures.

Fig. 106.—Gastroplasty.

Gastro-gastrostomy

Gastro-gastrostomy, an operation first employed in cases of hourglass stomach by Wölfler in 1894, consists in making a lateral anastomosis between the adjacent parts of the gastric pouches (Fig. 107). Two rubber covered clamps are applied to the stomach, one in the cardiac, the

other in the pyloric pouch, lying parallel to each other and transverse to the long axis of the stomach. The usual technique of lateral anastomosis by suture is followed. The opening should be at least 8 cm.

FIG. 107.—Gastro-gastrostomy.

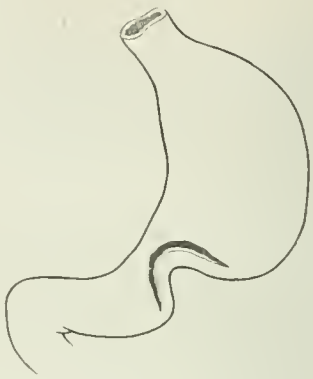


FIG. 108.—Gastro-anastomosis.

long. If the form of the constriction prevents so large an anastomosis, some other operation should be employed.

GASTRO-ANASTOMOSIS

Gastro-anastomosis.—This term may be used to designate an operation for hour-glass stomach analogous to Finney's pyloroplasty, introduced in 1903 by Kammerer. This differs from gastro-gastrostomy only in that the incisions meet, becoming continuous one with the other through the anterior wall of the channel connecting the two gastric pouches (Fig. 108).

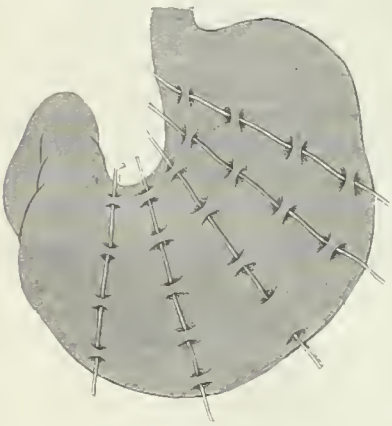


FIG. 109.—Gastro-plication.

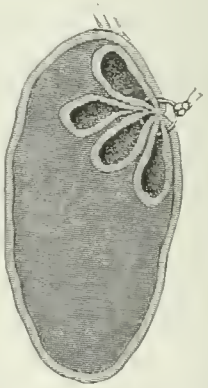


FIG. 110.—Gastro-plication, Seen in Sagittal Section.

Gastro-plication

Gastro-plication.—This operation, proposed in 1891 by Bircher, is usually done by Moynihan's modification of Bennett's method. Interrupted Lembert sutures are placed in the anterior gastric wall, each suture picking up this structure in four or five places, and running from the greater to the lesser curvature, transversely to the long axis of the stomach. As these sutures are tightened, the anterior wall of the stomach is puckered up, and the curvatures approach each other, thus diminishing the capacity of the stomach (Figs. 109 and 110).

Gastropexy

Gastropexy, an operation designed to fix a proptosed stomach, may be performed by either Duret's or Beyea's method.

(1) Duret's Method (1896). The abdomen is opened through the left rectus muscle, but the parietal peritoneum in the upper portion of the wound is not divided. By interrupted or continuous Lembert sutures of linen the anterior gastric wall is sutured to the parietal peritoneum of the epigastric region. The sutures should be inserted near the lesser curvature of the stomach, and should include not only the peritoneum of the abdominal wall but also the muscle and overlying fascia. They should not, however, pass through the skin, as it is desirable that they should remain permanently.

(2) Beyea's Method (1899). Interrupted sutures of linen are passed through the gastro-hepatic omentum from the stomach up to the under surface of the liver; each suture picks up the lesser omentum in four or five places. As these sutures are tightened the lesser curvature of the stomach is drawn up against the liver by the puckering of the gastro-hepatic omentum. Care should be taken not to puncture any blood-vessels in this structure.

Gastrectomy

Gastrectomy.—The terminology employed by writers with regard to excision of portions of the stomach is not always uniform, and unless the terms used are clearly defined confusion is liable to arise. In the present work we employ the following terms to designate the operations denoted below. *Sphincterectomy:* By this we understand the removal merely of the pyloric sphincter, with end-to-end reunion of the duodenum and the stomach. It is an operation which in a few rare instances has been employed for benign fibrous stenosis of the

pylorus, in which the pre-pyloric portion of the stomach was healthy. Pyloroplasty, we think, would be a less dangerous and quite as satisfactory an operation. By *Pylorectomy* we mean removal of the pylorus with its antrum (Fig. 7); it is a more extended resection than sphincterectomy, but less so than partial gastrectomy, in which latter operation the entire lesser curvature of the stomach is removed. In general, we mean by *partial gastrectomy* an operation which removes besides the pylorus, also the neighboring part of the stomach as far as the Hartmann or Mikulicz line, *always including the whole of the lesser curvature* (Fig. 111). If the gastric area removed extends still further towards the fundus along the greater curvature, we speak of the operation as *subtotal gastrectomy* (Fig. 112); while the term *total gastrectomy* is reserved for operations which leave behind no portion of the stomach, the upper section passing through the lower end of the esophagus, while the lower section of course divides the duodenum. *Circular* or *cylindrical gastrectomy* designates an operation by which the central portion of the stomach is removed, neither the pylorus nor the fundus being included in the section, although the lines of division extend from one curvature to the other and involve the entire circumference of the stomach (Fig. 120); in this operation the lumen of the stomach is restored by an end-to-end anastomosis (circular gastrorrhaphy). By *gastric resection* we understand removal of a portion of the stomach not including the entire lumen of the organ; the term *plastic resection* we think therefore properly describes the operation called gastroplasty by Jedlicka (1904); since in this operation, after resection of a portion of the anterior wall of the stomach, he reconstructed the organ by a plastic operation. *Excision* in connection with gastric operations we would limit to the removal of more or less circumscribed lesions or pedunculated tumors attached to or springing from the stomach (Plate I, p. 70).

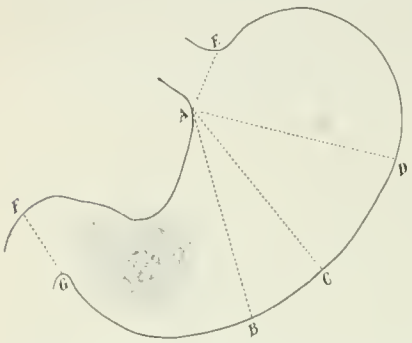


FIG. 111.—Diagram Showing Various Incisions for Gastrectomy. *F*, *G*, Duodenal Section; *AB*, Hartmann Line; *AC*, Mikulicz Line; *AD*, Mayo Line; *AE*, Total Gastrectomy. (*After Paterson*, 1906.)

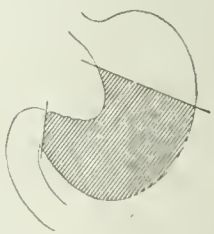


FIG. 112.—Diagram to Show Portion of Stomach Removed in Subtotal Gastrectomy.

As many of these operations are always *atypical* it is not possible to describe them in detail. It being well appreciated that every surgeon of experience develops a technique more or less peculiar to himself, and that all that can be asked in a work of this kind is an adequate description of certain *typical* operations, we have selected the following for discussion: I. Pylorectomy, by Billroth's first method. II. Partial Gastrectomy, including Kocher's method, Billroth's second method, and Polya's method. III. Total Gastrectomy. IV. Cylindrical Gastrectomy. V. Gastric Resection.

The **indications** for the employment of gastrectomy have been discussed in previous chapters (pp. 111, 276).

Pylorectomy by Billroth's First Method.—This operation (Fig. 113) was first performed by Péan in 1879, then by Rydygier in 1880, and first successfully by Billroth in 1881.

Open the abdomen by an incision 10 to 12 cm. long, close to the median line, through the right rectus muscle. Place sufficient gauze packs in the lower part of the wound to keep the small intestines and transverse colon from protruding. Identify the stomach, and doubly ligate the coronary artery on the lesser curvature at the site selected for the gastric section, and cut the artery between the ligatures. Doubly ligate the gastro-epiploic artery on the greater curvature at the other extremity of the proposed section, and divide it between the ligatures. Ligate in sections, by means of an aneurysm needle, the gastro-hepatic and the gastro-colic omenta, from the line of the proposed section in the stomach to the duodenum. At the greater curvature this row of ligatures should pass between the gastro-epiploic artery and the transverse colon, great care being exercised not to include the middle colic artery in any of the ligatures. The lesser omentum should be ligated fairly close to the liver.

FIG. 113.—Diagram of Billroth's First Method of Gastrectomy.

Pass a gastrectomy clamp, with rubber sheathed blades, from the greater to the lesser curvature, at the site of the proposed section of the stomach. Bring its points out far enough beyond the lesser curvature to grasp firmly between the blades the whole of the proposed section of the stomach. Parallel to this first clamp pass a second on the pyloric side of the gastric section, about one inch distant from the first clamp. The latter clamp need not have its blades covered with rubber. Pass a hot moist sterile gauze pack across the lesser peritoneal

cavity from one curvature of the stomach to the other, immediately beneath the two clamps already placed. Then divide the stomach with the scalpel or the actual cautery from one curvature to the other, between the two clamps, but close to the clamp on the pyloric side of the section, so as to leave enough tissue protruding from the rubber covered clamp to make the application of sutures easy. Turn the pyloric portion of the stomach thus set free over to the patient's right, and place sufficient gauze behind the stomach and pylorus to protect thoroughly the lesser peritoneal cavity. Having thus mobilized the pyloric portion of the stomach, it will be easy to detect the pyloric artery on the lesser curvature, and the beginning of the right gastro-epiploic artery on the greater, just below the pylorus. It is not necessary to ligate the gastro-duodenal artery above the pylorus; to do so might impair the blood supply to the descending duodenum and the head of the pancreas. When the pyloric and right gastro-epiploic arteries have been ligated, a rubber covered clamp is to be applied to the first part of the duodenum, and the pyloric part of the proposed duodenal section is to be guarded by another clamp. Close to this latter the duodenum is next divided by scalpel or actual cautery, and the diseased part of the stomach including the pylorus is removed.

The gastric segment will now present a much larger area than the duodenal. Hence the surgeon should begin his through-and-through sutures at the lesser curvature of the gastric segment, and close this portion of the stomach from above downward until the unsutured portion presents the same calibre as does the duodenum. The latter is then drawn across to the gastric segment and united to its unsutured portion first by a posterior sero-serous continuous suture of linen; then the through-and-through (chromic gut) suture is resumed, and the duodenum united to the stomach throughout the circumference of the bowel, great care being exercised to secure accurate approximation at the point where the suture line of the gastric section meets the gastro-duodenal anastomosis. This point of junction is known as the "deadly angle" from the frequency with which leakage has occurred there (Fig. 113). When the duodenum and stomach are thus united securely by a through-and-through suture, the gastric and duodenal clamps are removed, and any bleeding points reinforced by specially inserted sutures. Finally the entire line of sutures, both that of the gastric segment and that of the anastomosis, should be inverted by a continuous sero-serous suture. The gauze packs may now be removed; the remains of the gastro-hepatic and gastrocolic omenta are stitched to the upper

and lower portions of the anastomosis, and the abdominal wound is closed.

Partial Gastrectomy. (1) **Kocher's Method** (1891).—In this operation the distal segment of the divided duodenum is implanted (termino-lateral anastomosis) into the posterior wall of the stomach, a little to the left of the gastric section which is entirely closed (Fig. 114). Kocher used crushing forceps in this operation, instead of the rubber covered clamps employed by most surgeons; he made the section of the stomach close to the crushing forceps, and as a consequence it was necessary for the through-and-through sutures to be applied on the cardiac side of the crushing forceps. Then when these forceps are removed, the projecting tissue is trimmed close to the line of the through-and-through sutures, and the latter are inverted by a running sero-serous suture. The following description of the operation is taken from the English translation by Stiles of Kocher's Operative Surgery (London, 1903, p. 215).

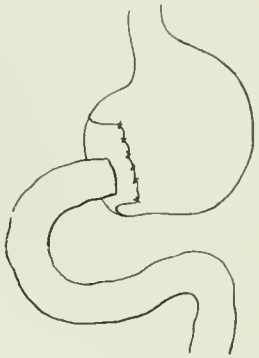


FIG. 114.—Diagram of Kocher's Method of Gastrectomy.

"After ascertaining exactly the limits of the tumor and the mobility and the possibility of separating glands, the lesser omentum and the gastrocolic ligament are perforated at the margin of the new growth towards the fundus of the stomach, and two large pressure-forceps are applied quite close to each other, and closed as firmly as possible. After gauze pads have been placed beneath the forceps (the aseptic protecting pads have previously been placed round the parts outside the abdomen) the stomach is cut across between the two clamps, close up to the one to the right. According to Hartmann's rule, and on the grounds of Cunéo's observations, the clamps must be applied to the lesser curvature as high up and as much to the left as possible; and in order that the glands that accompany the coronary vessels may at the same time be removed, it appears to us advisable to double ligature and cut across the coronary artery above the point where the section is to be made. By dividing bloodlessly the small omentum above the glands the divided stomach is rendered so movable that it can be turned over to the right side.

"Hartmann places value on the last procedure because the fatty tissue and glands can then be followed up along the lesser curvature and can be included in the removal as far as the origin of the pyloric and the gastroduodenal branches of the hepatic artery. The latter

vessel is carefully avoided, while the two first named are ligatured. After throwing the stomach over towards the right margin of the wound, one sees quite well the gastroduodenal artery running downwards in the groove between the duodenum and pancreas. In this way no bleeding should occur in detaching the chain of glands which accompany this artery along the above-mentioned groove. We can manage quite well without ligaturing the artery. Throwing the stomach over to the right edge of the wound has the advantge that by drawing upon it the duodenum is rendered quite accessible from behind, so that the limits of the new growth can be ascertained with certainty, and one can determine if the duodenum be long enough and movable enough to enable one to perform gastroduodenostomy. When this is the case, two small pressure-forceps are now applied to the duodenum, which is cut across between them with the knife close up to the pair farther removed from the stomach, a small pad of gauze having previously been placed under the part. The cut edges are then carefully and thoroughly cleansed.

"The opening can now be made into the stomach before closing it with sutures, because the forceps in position afford a very good support. A pair of clamp-forceps is applied to the duodenum and the accompanying vessels, and the crushing-forceps are removed. The fingers of the assistant can now grasp the stomach so that the anterior wall is pressed against the posterior wall at the place where the incision has been made; or a pair of clamp-forceps may be applied to the stomach after it has been closed by suturing and before the incision is made for anastomosis with the duodenum.

"A continuous mattress suture, after the manner of a half Gély's suture, is carried behind the crushing-forceps (which has not been removed from the stomach); only the commencement is knotted, and by pulling on the two ends reliable closure is effected. If the crushing-forceps are not strong enough to compress the tissues so that they are like thin dry paper, it is desirable that every projecting portion of mucous membrane and muscular pulp should be clipped away with scissors, but this is unnecessary if sufficiently powerful compression (crushing) forceps are employed. With the ends of the suture held taut, a continuous glover's suture is rapidly applied over it so as to fix each loop, and the closure is completed by inverting both by a continuous serous suture.

"The stomach and duodenum are now clamped at some distance from the place where they have been opened, or are to be opened, and the crushing forceps are removed from the duodenum. Escape of

the contents being carefully prevented, an incision is made into the posterior wall of the stomach near the greater curvature at a distance of 3 cm. (about one inch and a quarter) from, and parallel to, the sutures which close it, at first merely through the serosa, for a length equal to the breadth of the duodenum. The posterior segment of the circular suture is now applied. The wall of the stomach is then completely cut through and a continuous suture, passing through all the coats, is applied so as to unite the posterior edges of the opening in the stomach and duodenum. A third continuous suture is applied so as to unite the two mucous edges. The anterior edges are now united by a continuous suture which passes through all the coats, and is knotted at each end to the posterior suture. The clamp-forceps, if such have been employed, are removed, and lastly, the anterior serous suture is applied and knotted at each end with the posterior serous suture."

Remarks.—The technique employed by Kocher, as is seen from the above description, coincides with that preferred by Hartmann, in that the stomach is divided before the duodenum, is then turned to the patient's right, and the duodenal section made after clearing the glands away from along the gastro-duodenal artery. The particular part of the operation by virtue of which Kocher's name has been attached to it, consists in the implantation of the duodenum into the posterior wall of the stomach. If the removal of the stomach has been at all extensive, it is very likely that it will be impossible to bring the duodenum and stomach into apposition, even after mobilizing the duodenum. In such circumstances gastro-jejunostomy should be done.

(2) **Billroth's Second Method.**—This method, in which the operation is terminated by a gastro-jejunostomy, is that which is commonly performed. Usually the anastomosis should be made in the posterior wall of the stomach; but if the patient's condition is bad, or if the remaining portion of the stomach is very small, anterior gastro-jejunostomy, as done by Billroth himself, may be adopted. The use of a Murphy button may hasten the procedure in desperate cases.

The technique habitually employed is the following:

After exposing the stomach, the coronary artery is identified, doubly ligated and divided, close to the cardiac orifice of the stomach. The finger is passed through the gastro-hepatic omentum into the lesser peritoneal cavity, and the gastro-hepatic omentum is ligated in sections, fairly close to the transverse fissure of the liver. By cutting through the gastro-hepatic omentum, the surgeon reaches the pyloric artery, which is doubly ligated and cut. The finger is then passed

down behind the pylorus, and the right gastro-epiploic artery is identified below the pylorus; this artery is ligated in two places, and cut between the ligatures. Hemostatic forceps are then applied to the gastrocolic omentum, and as they are applied this structure is divided between them, beginning at the pylorus and passing along the upper border of the transverse colon until the point is reached at which it is proposed to divide the stomach. This point should be 5 cm. to

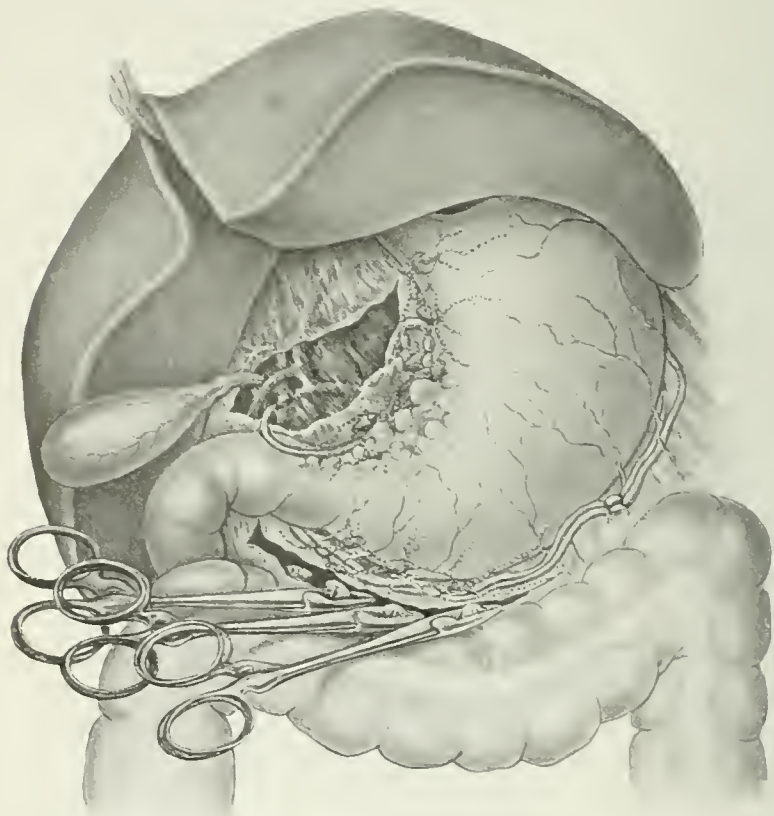


Fig. 115.—Partial Gastrectomy: Division of Gastro-colic Omentum.

the left of the visible malignant growth. When this point has been reached, the left gastroepiploic artery is ligated just to the left of the proposed gastric incision. In placing the hemostats on the gastrocolic omentum, great care is to be taken to avoid the middle colic artery and its branches (Fig. 115).

The portion of stomach to be removed is now completely freed along its curvatures, and remains attached only to the duodenum and

the body of the stomach. The lesser peritoneal cavity can now be protected thoroughly by sterile gauze compresses. A clamp with rubber-covered blades is now applied to the duodenum about 2.5 cm. beyond the portion visibly diseased, and an ordinary clamp is applied just to the pyloric side of the first clamp. The duodenum is then divided between the two. The entire portion of the stomach to be excised can now be turned to the patient's left. The duodenal stump is closed first by a through-and-through chromic catgut suture; before the occluding clamp is removed a purse-string suture of linen

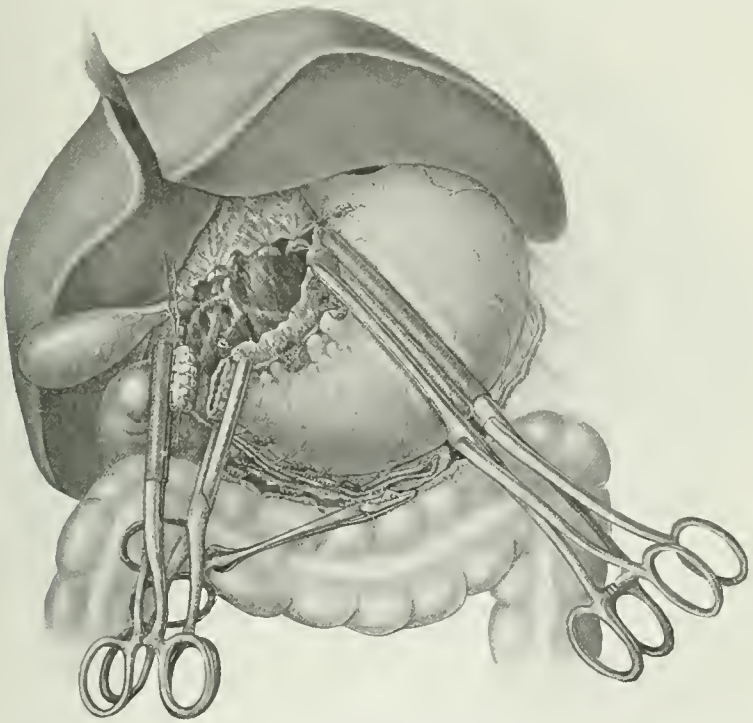


FIG. 116.—Partial Gastrectomy: The Duodenum has been Divided, and the Clamps are in Place for the Gastric Section.

is applied; the clamp is then removed, and by catching the duodenal wall in two places with dissecting forceps, the sutured end of the duodenum is inverted and the purse-string suture is drawn tight and tied. Sometimes a few additional Lembert sutures of linen are inserted to re-inforce those previously placed. As the duodenum is divided where part of its wall is retroperitoneal, it is very important to suture it accurately; but usually the through-and-through suture and the purse-string suture are all that is necessary. The gastro-colic omentum is then ligated, and the hemostatic forceps removed.

Rubber-covered gastrectomy clamps are then applied across the stomach from the greater to the lesser curvature, at least 5 cm. to the left of the visible malignant growth (Fig. 116). Clamps with a screw lock at the end of the blades, as in Kocher's clamps, are safest. The entire lesser curvature is always removed, but the line of the section passing from that point to the greater curvature varies with the extent of the tumor. About 2 cm. to the patient's right of this occluding clamp, an ordinary forceps is applied, and the stomach is divided between the two with the actual cautery. The excised portion being removed, a

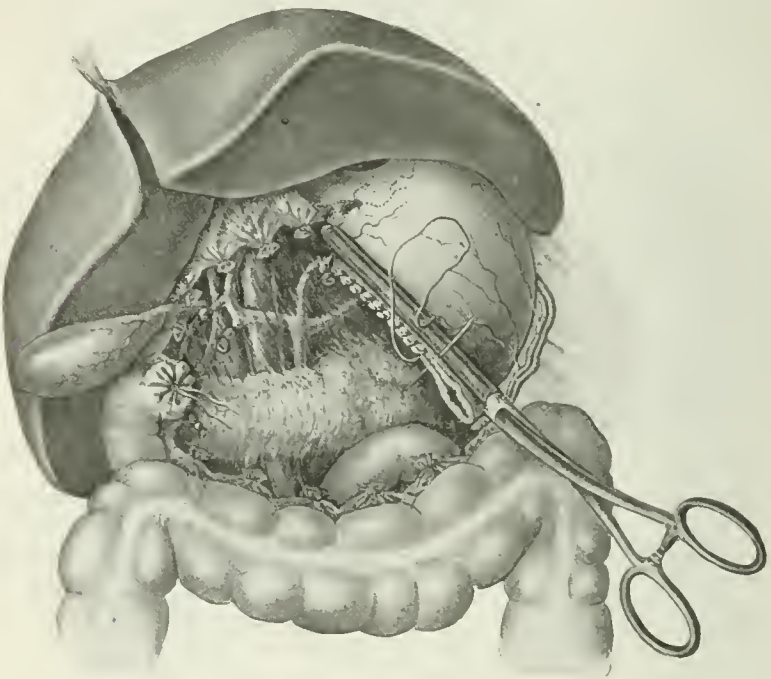


FIG. 117.—Partial Gastrectomy: Through-and-through Sutures being Applied to the Gastric Section.

through-and-through suture of chromic catgut is inserted through the margins of the gastric walls which protrude from between the blades of the rubber-covered clamp (Fig. 117). It is well to grasp these margins at one or more points with forceps to prevent their retracting. When the through-and-through sutures have been completed, the clamp is removed, and a continuous sero-serous suture is applied, burying the first row, and carefully re-inforcing any points that tend to bleed (Fig. 118).

The transverse colon is then drawn out of the wound, and the

posterior gastric wall is exposed by opening the transverse mesocolon. A posterior gastro-jejunostomy is then done by the usual technique, as described at page 338, and as indicated diagrammatically in Fig. 118.

Finally, after suturing the margins of the opening in the transverse mesocolon to the gastric wall, and replacing the newly formed anastomosis within the abdomen, the great omentum is drawn up to cover the space left by the removal of the stomach, and the abdominal wound is closed.

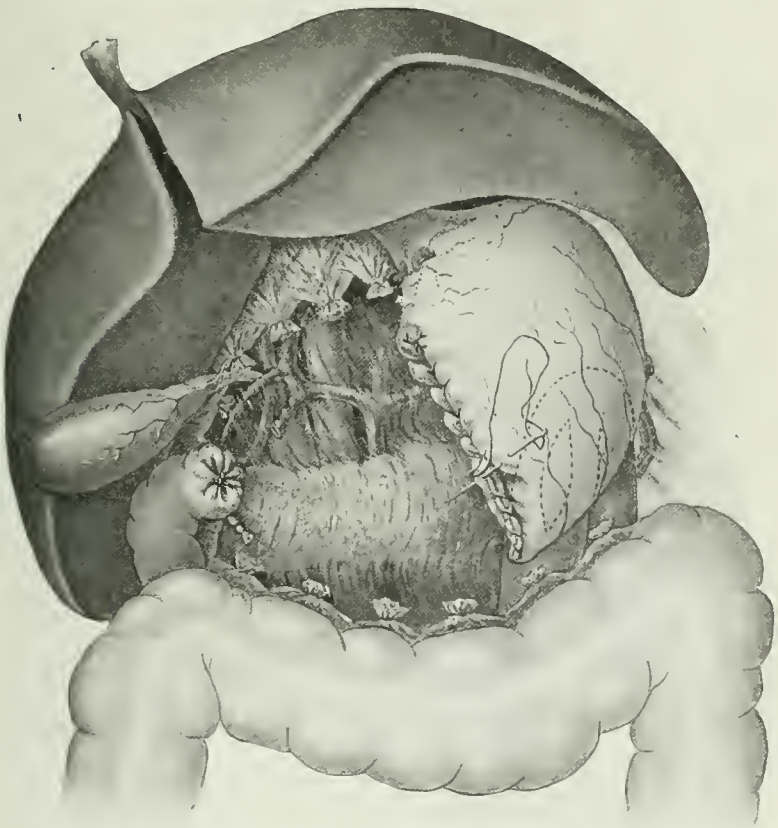


FIG. 118.—Partial Gastrectomy: Sero-serous Sutures being Applied to the Stomach.

(3) **Polya's Method** (1911).—In this method the opened end of the stomach left after removal of the diseased portion is implanted into the side of the jejunum, which is drawn up through the transverse mesocolon.[1] The stomach is exposed in the usual way, the gastro-hepatic

[1]Mikulicz stated as early as 1898 that he preferred this method of completing the operation of gastrectomy; but credit is undoubtedly due to Polya, and in this country to Mayo, for standardizing and popularizing the procedure.

and gastro-colic omenta are divided, the duodenum sectioned and closed and the stomach is turned to the patient's left. An antero-posterior incision is then made in an avascular area of the transverse meso-colon from its upper surface, and the first coil of jejunum is drawn up from below through this opening (Fig. 119). It is next attached, by a continuous

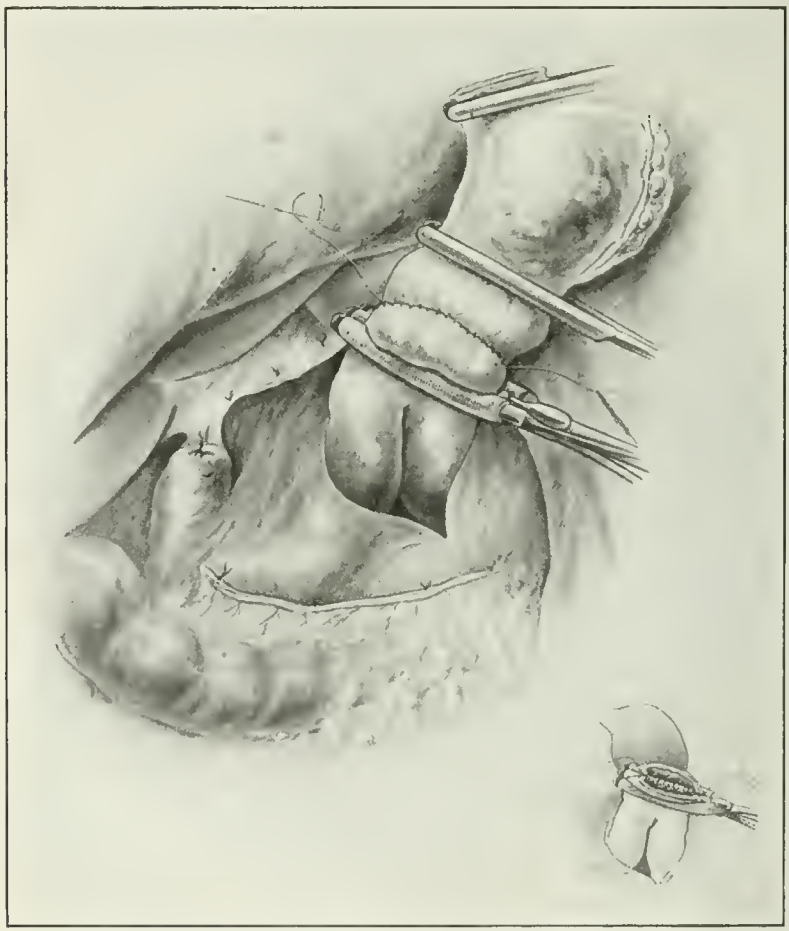


FIG. 119.—Polya's Method of Gastrectomy: After Section and Closure of the Duodenum, the Stomach is Turned Upward to the Patient's Left, Fully Exposing Its Posterior Wall; the Transverse Mesocolon is Opened, the First Coil of the Jejunum is Drawn Through It, and Anastomosed with the Sectioned End of the Stomach, as Indicated in the Smaller Drawing. From a Dissection by the Junior Author in the Laboratory of Operative Surgery, University of Pennsylvania.

sero-serous suture of linen, to the posterior wall of the stomach, well beyond the diseased area, in such a way that the proximal portion of jejunum corresponds to the lesser curvature, and the distal portion of the jejunum to the greater curvature of the stomach. Before cutting

away the stomach an occluding clamp should be applied on each side of the proposed section. The jejunum also is clamped before it is opened (Fig. 119), along its antimesenteric border for a distance nearly equal to the opened end of the stomach, which is to be implanted into the side of the jejunum. The anastomosis between stomach and jejunum is then completed in the ordinary manner, the clamps are removed, the margins of the opening in the mesocolon are sutured to the stomach above the anastomosis, and the abdominal wound is closed in the usual way.

This is an excellent operation, presenting distinct advantages not possessed by Billroth's second method. Time is saved in that a separate anastomosis is not necessary after the cut end of the stomach has been disposed of; less exposure is required of the abdominal contents as it is not necessary to deliver the great omentum and transverse colon from the abdominal incision in order to expose the jejunum and do a posterior gastrojejunostomy; and the restoration of the gastrointestinal canal is much simpler and easier in cases of extensive removal of the stomach.

In 1917 Mayo and Balfour adopted an antecolic anastomosis between jejunum and stomach by Polya's technique. This presents certain obvious advantages in cases of subtotal gastrectomy. They prefer to close the sectioned end of the stomach for a convenient distance upward from the greater curvature, thus making the anastomotic opening into the jejunum comparatively small and close to the lesser curvature of the stomach.

Subtotal gastrectomy differs from partial gastrectomy only in the extent of the stomach removed. A precisely similar technique may be employed. In some cases, however, there will be so small a portion of the cardia left that only an anterior gastro-jejunostomy can be done. As already remarked, Mayo and Balfour's modification (1917) of Polya's technique is suitable for such cases.

Remarks.—It is seen that the technique of partial gastrectomy as above given is practically identical with that described by Mayo in 1904. Although the technique of Hartmann, in which the gastric section is made first, and the tumor then turned to the patient's right, before dividing the duodenum, presents the undoubted advantage of approaching the dangerous retro-pyloric region in an open and strictly anatomical way, yet it has the disadvantage, as pointed out by Mayo, of being less easy of accomplishment than the method in which the duodenum is divided first; because the line of proposed section of the stomach is frequently difficult of access until the stomach is mobilized

by section of the duodenum. We would point out, moreover, that Mayo's operation has the distinct advantage that the occluding clamps are not applied to the stomach until the last possible moment, and that they are kept in place for the very shortest possible time—in fact, only until it is possible to insert the through-and-through sutures. There is thus scarcely any risk of interference with the vascular supply of the sutured edges.

Total Gastrectomy.—In rare cases after the abdomen has been opened it may be found that the disease has invaded so great an area of the stomach as to render its entire removal desirable, while at the same time no secondary growths or adhesions exist which will render such an operation impracticable.

The operation should proceed along the same lines as partial gastrectomy, until the duodenum has been divided. It is then to be determined whether the duodenum can be made to reach the esophagus without undue tension. Mobilization of the duodenum, as in operations on the retro-duodenal portion of the common bile duct, may render this possible. If the duodenum can be made to reach the esophagus, it should be attached to the latter by a primary posterior row of sutures before the cardiac orifice of the stomach is divided. Exposure of the cardia as in Lambert's method (Figs. 44, 45) may facilitate the subsequent steps. Then the cardia is clamped, divided above the clamp, and the union of the duodenum to the esophagus completed, an end-to-end anastomosis being performed in the usual way. If the duodenum cannot be made to reach the esophagus, a coil of the upper jejunum, provided with a long mesentery, should be selected; the jejunum should be divided completely across, its distal end being united to the esophagus by circular (end-to-end) anastomosis, and the proximal end being implanted into the distal segment at a convenient distance below the esophago-jejunal anastomosis. The jejunum should be united to the esophagus by the trans-mesocolic route if possible. The Murphy button may be employed if accurate suturing is impossible.

Should the surgeon be so heedless as to remove the entire stomach before determining whether any portion of the intestinal tract can be anastomosed to the esophagus, he should insert a tube into the divided end of the duodenum and suture the latter into the abdominal wound; should this be impossible, jejunostomy may be a last resort.

It might be possible to connect the esophagus and duodenum by an excluded loop of the jejunum, transplanted through the transverse mesocolon, somewhat after the manner of Herzen's operation of gastrostomy (p. 324).

Vassalo reported (1906) a case of total gastrectomy in which the entire time consumed in the operation was only thirty-eight minutes.

Cylindrical Gastrectomy

Cylindrical Gastrectomy.—This operation (known also as "Sleeve-resection") is suitable only for benign lesions occupying the middle zone of the stomach (Figs. 120 to 123). If there exist an ulcerated area which does not obstruct either orifice of the stomach, as is the case with some ulcers along the lesser curvature; and if some form of radical operation is to be preferred to gastro-jejunostomy, then a cylindrical

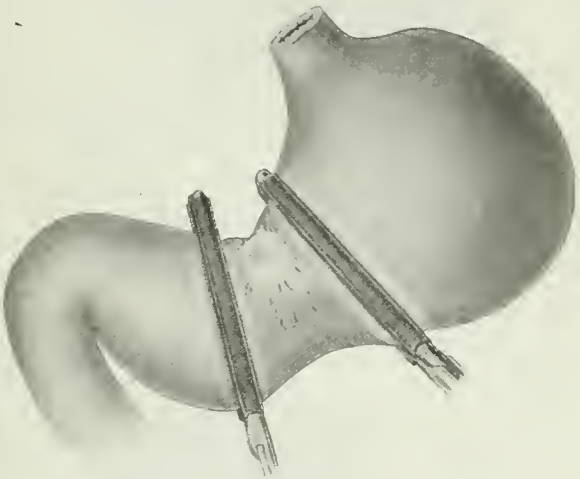


FIG. 120.—Cylindrical Gastrectomy, the Clamps in Place.

gastrectomy frequently will be simpler and easier of accomplishment than resection of a V-shaped area from the lesser curvature. In some cases of hour-glass stomach cylindrical gastrectomy may be of benefit.

We entirely agree with Leriche (1907), however, in his dictum that cylindrical gastrectomy is absolutely contra-indicated if there are enlarged glands in the gastro-hepatic omentum; but we would go further, and prohibit its employment in *every* case of malignant disease, because under such circumstances the entire lesser curvature should be removed. Leriche, who is a supporter of excision of benign lesions in general, makes the bold but possibly significant statement that if this operation were more often done for non-stenosing cancer, more cases of progressive pernicious anemia would be cured.

The operation may be performed thus: After exposing the stomach and ligating the main arteries at the extremities of the proposed

sections, the gastro-colic and gastro-hepatic omenta are next ligated, and divided. Then two pairs of rubber-covered clamps are applied,

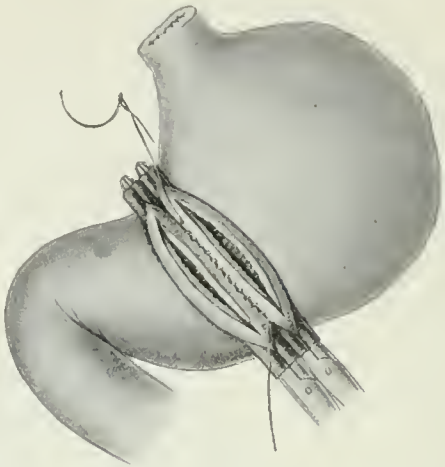


FIG. 121.—Cylindrical Gastrectomy: After Removal of the Diseased Area, the Portions of the Stomach Remaining are United by End-to-end Anastomosis.

including between them the portion of stomach to be removed, which should be emptied as completely as possible before tightening the

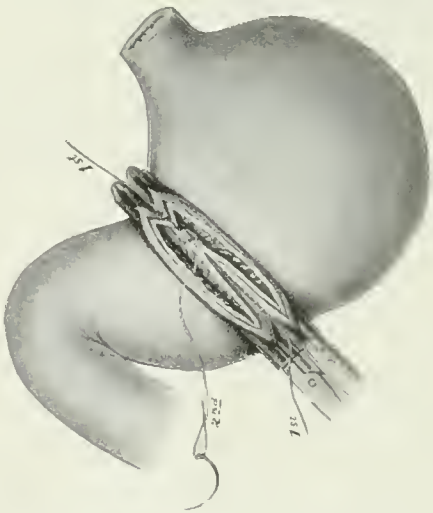


FIG. 122.—Cylindrical Gastrectomy: The Through-and-through Suture for End-to-end Anastomosis being Applied.

clamps. The diseased portion of the stomach is then cut away, and the divided surfaces of the stomach united by circular gastrorrhaphy

(end-to-end anastomosis). Leriche calls attention to the advantages of commencing and terminating the continuous sutures, not at one of the curvatures of the stomach, where the peritoneal coat is defective, but at some convenient point on the anterior gastric wall. If the two portions of the stomach do not meet with the utmost facility, that is to say, if there be the very least tension on the sutures anywhere, it will be possible to overcome this by *mobilization of the duodenum*. In

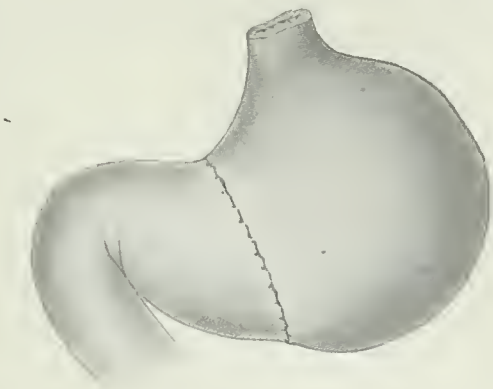


FIG. 123.—Cylindrical Gastrectomy. The Operation Completed.

some cases it may seem better to terminate the operation as in *exclusion of the pylorus*, by closing both gastric segments and doing a separate gastro-jejunostomy.

Hartmann (1914) objected to cylindrical gastrectomy that it interfered markedly with peristalsis; but G. D. Stewart and Barber (1916) concluded that there was not much if any delay in evacuation of such stomachs; and that in these respects it was a more satisfactory operation than gastric resection.

GASTRIC RESECTION

Gastric Resection.—Removal of a *wedge-shaped area* from the lesser curvature of the stomach may be accomplished by applying rubber-covered clamps outside of the proposed lines of section, after tying off the gastro-hepatic omentum. The wounds left may then be sutured the one to the other, thus approximating the cardiac and pyloric orifices. This mode of reunion may prove difficult or even impossible; under which circumstances a complicated form of plastic operation may have to be undertaken. This operation, therefore,

is not one to be lightly undertaken; indeed we cannot see that it presents any advantages over cylindrical gastrectomy.

In many cases resection of a callous ulcer must be *atypical;* but always, in closing the defect, an effort should be made not to cause stenosis of the gastric canal, but rather to enlarge it by suturing the defect transversely to the long axis of the stomach.

Jedlicka adopted (1904) a form of *plastic resection* of the stomach which he calls *gastroplasty*. By this operation, after removing the diseased portion of the lesser curvature and of the anterior or posterior walls of the stomach, he reconstructs the natural contour of the stomach by the method of sliding flaps. In one case the operation took two hours and a quarter.

Excision of Ulcers on the Posterior Wall.—If the ulcer is close to either curvature, it usually may be exposed through the corresponding omentum (gastro-hepatic or gastro-colic).

If not readily accessible in this way, the surgeon may adopt the *inter-colo-epiploic route*, as it is called, detaching bloodlessly the great omentum from the transverse colon, displacing the former upward and the latter downward. This route, which was particularly studied by Lardennois and Okinczyc (1913) in connection with colectomy, was adopted by Pauchet (1916) for repair of gunshot wounds involving the the posterior gastric wall, and for exploration of the posterior wall in cases of suspected ulcer and for excision of ulcers adherent to the pancreas. Fig. 11 indicates what ample exposure may be gained, not only of the body of the stomach, but even of the posterior surface of the pylorus and duodenum. The bloodless area between the epiploic branches of the gastro-epiploic arteries above and the bloodvessels of the transverse mesocolon below is best identified by pulling the omentum out of the wound until the transverse colon becomes taut; then the dissection commences toward the flexures of the colon and approaches the centre from both sides.

Transgastric Excision.—It may be simpler to open the anterior wall of the stomach by an ample incision and to excise the ulcer from within, as indicated in Fig. 124. This plan was adopted by Pilcher in 1907, and popularized by Mayo in 1910. After repair of the defect from the interior of the stomach by continuous suture of chromic gut, the serous surface of the wound in the posterior wall may be exposed through a comparatively small opening in the gastro-colic omentum by the aid of the fingers in the cavity of the stomach; and a reinforcing suture of linen applied. Mayo considers a single row of through-and-through sutures, applied from the mucous surface, sufficient in most

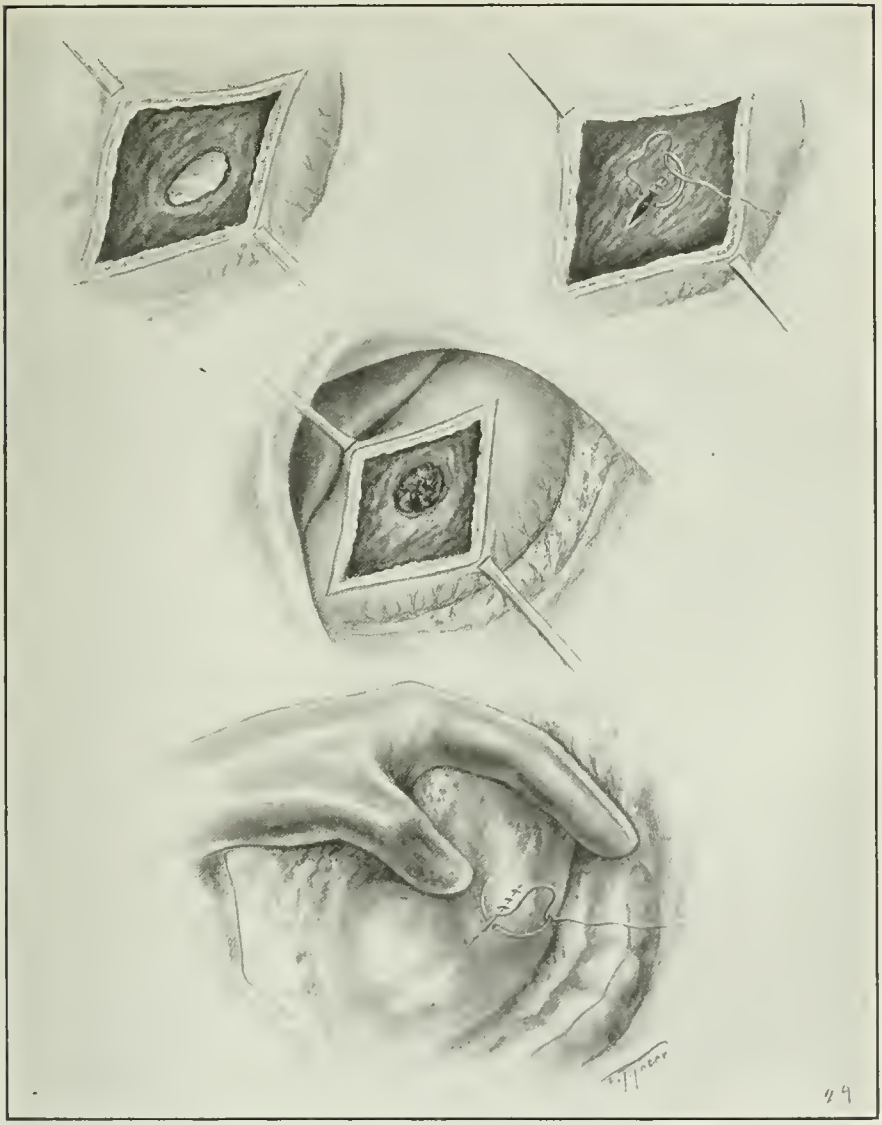


FIG. 124.—Transgastric Excision of an Ulcer on the Posterior Wall of the Stomach: In the Centre the Ulcer is Exposed; Above it is Shown Excised, and Sutured from Within; Below a Reinforcing Sero-serous Suture is Being Applied after Exposing the Posterior Wall Through the Gastrocolic Omentum. From a Dissection by the Junior Author in the Laboratory of Operative Surgery, University of Pennsylvania.

cases. Then the opening in the stomach's anterior wall is closed in the usual manner.

Resection of the cardia, founded on the advances in thoracic surgery inaugurated by Sauerbruch (1905) and by Brauer, was accomplished in one case of carcinoma by Wendel (1907) with the use of Brauer's positive pressure air chamber around the patient's head. He operated on Sept. 6, 1906, making an incision 16 cm. long in the sixth left costal interspace, cutting through the seventh costal cartilage. Some adhesions between the lung and pleura were separated, and the lung was excluded from the field of operation by the use of gauze packs. The vagi were then freed without difficulty, the lower end of the esophagus isolated, and gauze was passed behind it. The esophagus was then freed from the diaphragm. This work was absolutely in the dark, and accomplished only by the sense of touch; it was very difficult; there was free arterial bleeding, and the bleeding points were very difficult to ligate. The time consumed was now an hour and a half. It was found impossible to do an anastomosis between the esophagus and the stomach to one side of the tumor, so resection was undertaken. The tumor could be drawn out through the diaphragm and across the pleural cavity, even to the thoracic wound. It was surrounded by gauze packs. The vagi were found entering the tumor and could not be dissected free lower down. They were therefore cut off 1 cm. from their point of entrance into the tumor. The esophagus was next divided, and its end closed by sutures. Before this was done, however, the male half of a Murphy button was inserted into the esophagus, and later was liberated by making a slit in the side of the latter. The gastric incision included practically all of the lesser curvature, and a good deal of the fundus of the stomach. The female half of the button was passed into the cavity of the stomach through the gastric wound, which was then completely closed, the shank of the button being made to protrude at another point of the gastric wall, where it was exposed by a puncture, and the two halves of the button approximated. Finally the margins of the diaphragmatic incision were sutured to the stomach below the anastomosis, and the intercostal wound was closed, with a strip of gauze for drainage. The time of the entire operation was somewhat over two hours. Although the patient reacted well, death occurred suddenly the next morning from secondary hemorrhage. The bleeding was found to come not from the line of sutures, but probably from one of the vessels which had been so difficult to ligate.

A similar operation had been done previously, and with success, several times on dogs, by Sauerbruch (1905) and by Sencert (1905) and

it had been attempted in two cases by Sauerbruch (1906) on man, but the patient in whom resection was attempted died on the table, and in the other the tumor was found to be inoperable, and an esophago-gastric anastomosis was done, the patient dying in 24 hours.

Wiener, in a patient with carcinoma of the lesser curvature of the stomach invading the cardia, employed osteo-plastic resection of the costal arch, under gas and ether anesthesia, and thirty days later, under spinal anesthesia, excised the growth, doing esophago-gastrostomy. Death occurred in eleven days, and was found to be due to a subphrenic abscess occasioned by separation of the esophago-gastric anastomosis. Wiener suggests doing a cervical esophagostomy of the lower end of the esophagus, and a gastrostomy, instead of attempting to unite the esophagus and stomach. Lambert's method (1914) of exposing the cardia, after section of the left coronary ligament of the liver, was mentioned at p. 176 (Figs. 44, 45).

W. Meyer (1915) collected 15 additional operations involving resection of the cardia, including two by himself. Three of the patients recovered and were doing well when reported several weeks after operation; a few others survived some days or weeks; but the majority died almost immediately. Meyer thinks an operation in several stages may ultimately become standardized for advanced cases: (*a*) *gastrostomy*, with careful exploration of the cardia; (*b*) excision of the tumor; (*c*) establishment of an esophageal fistula in the neck or over the upper sternum. H. Brun (1913), for operations on the cardia prefers the trans-thoracic route: he first mobilizes the duodenum and stomach and establishes a gastric fistula near the pylorus; at the second stage of the operation he opens the thorax and delivers the previously mobilized stomach through the diaphragm, when its excision becomes comparatively easy.

Excision of Descending Duodenum

Excision of Descending Duodenum.—While it may be impossible to excise the head of the pancreas without sacrificing the duodenum, on account of its blood supply (Sauvé, 1908; see p. 795), the reverse is not the case, according to Cotte and Maurizot (1910). These authors point out that the duodenum and pancreas do not present the intimate anatomical union which classical anatomy has taught. Their apparent union is due largely to the peritoneal reflections developed during intra-uterine life, and these may be separated, restoring the primary mobility of the duodenum.

Resection of the duodenum may be necessary for tumors of the

ampulla of Vater (see p. 591), or for pyloric tumors extending extraordinarily far into the duodenum; or rarely for carcinoma primary in the duodenum.

(*a*) If the duodenum alone is involved, the pylorus being healthy, the operator should begin by freeing the hepatic flexure of the colon by an incision through the parietal peritoneum on its outer side, and by dividing the cholecysto-colic fold. The gastrohepatic omentum is then opened above the first part of the duodenum, and the latter is depressed. The duodenum is then clamped, above, to the right of the pyloric vessels; and, below, to the right of the gastro-epiploica dextra. The structures of the hepatic pedicle (hepatic artery, portal vein, common bile-duct) are next isolated from above downward, clamping and tying the few arterial twigs running from the pancreas to the duodenum. If necessary the main trunk of the gastro-duodenal artery may be ligated, as in Hartmann's method of gastrectomy. By now pulling on the freed upper end of the duodenum, the peritoneal layers and fasciæ which unite the pancreas to the duodenum are made tense: the anterior layer is sectioned from above downward by scalpel, and then with curved scissors the pancreas and duodenum are cautiously separated, clamping and tying the few vessels which remain. The posterior pancreatico-duodenal layer of fascia will be found so attenuated that the separation may easily be accomplished by the finger. Below the region of the ampulla of Vater the separation of the pancreas from the duodenum is relatively easy. Finally the duodenum is sectioned just to the right of the superior mesenteric vessels; the *pancreatico-duodenalis inferior* artery lies behind the pancreas and is not apt to be injured. After section of the common duct and the duct of Wirsung, the duodenum is entirely free. It remains, of course, to restore an outlet for the stomach by some form of gastro-jejunostomy and to provide for the implantation of the bile and pancreatic ducts into the gastro-intestinal tract. (For Kausch's method, see p. 591.)

(*b*) In resections of the duodenum for pyloric growths, Hartmann's technique for partial gastrectomy should be followed (p. 353), sectioning first the body of the stomach and turning the tumor mass toward the patient's right, approaching the pylorus from the rear. Then mobilize the descending duodenum (p. 784), and return to the retropyloric region, opening by blunt dissection the layer of peritoneum which passes from the pancreas to the posterior wall of the duodenum. The gastro-duodenal and pyloric arteries are now tied and cut, whereupon the pylorus, being freed from all posterior attachments, may be drawn into the wound, perhaps far enough to permit division of the duodenum

well beyond the tumor. If necessary it can be exposed to a still lower level by pulling on the pyloric mass, thus opening up the posterior duodeno-pancreatic angle. To go further one must cut the peritoneum uniting the upper border and anterior surface of the duodenum and pancreas; and as their posterior surfaces have already been detached, the remainder is quite easy: all the main arterial trunks having already been tied, there is a mere ooze. The closure of the stump of duodenum after section becomes as simple as the closure of any other coil of small intestine.

JEJUNOSTOMY

Jejunostomy.—This operation was first employed in 1878 by Surmay, of Ham, in the case of a patient with carcinoma; death occurred the next day, from peritonitis.

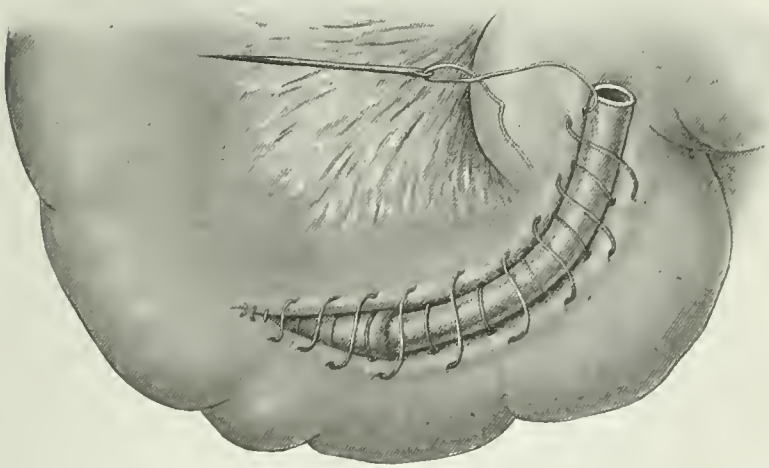


FIG. 125.—Jejunostomy by the Method of Karewski.

Karewski (1896) adopted the *method of Witzel* for gastrostomy (1891). A coil of the jejunum about 45 cm. from its origin is selected, and the catheter is sutured in place, as shown in Fig. 125, with its eye end toward the anal end of the bowel. Then the bowel is attached to the parietal peritoneum at the edges of the abdominal incision, and the latter is sutured close up to the tube. Feeding should be begun at once.

In Maydl's operation (1898) the jejunum is completely divided about 20 cm. below its origin, the proximal segment is implanted (end-to-side anastomosis) into the distal about 20 to 30 cm. below the

section, and the distal segment is sutured end-on into the abdominal wound (Fig. 126).

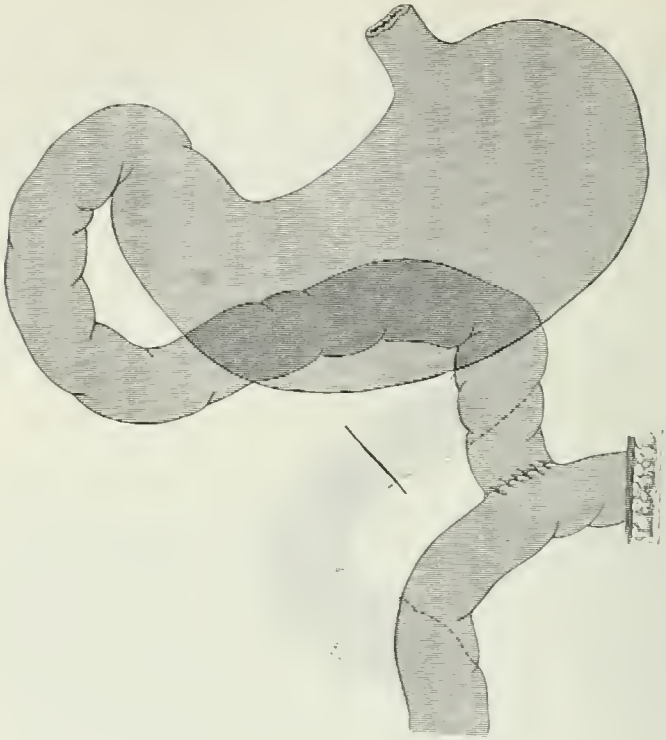


FIG. 126.—Jejunostomy in-Y. Method of Maydl.

Duodenostomy, in which the fistula is made (after the method of Witzel and Karewski) in the duodenum above the bile papilla, is preferred to jejunostomy by Hartmann (1903).

CHAPTER XV

COMPLICATIONS AND SEQUELS OF OPERATIONS ON THE STOMACH AND DUODENUM

The operations which are now employed in the treatment of gastric diseases are much less frequently followed by untoward symptoms than was the case in the earlier periods of the surgery of the stomach. Yet certain complications and sequels still demand attentive study by the surgeon, because even now a patient is occasionally seen who has been operated on by an antiquated method, and who presents the symptoms of one of the sequels formerly not unusual. Moreover, there are certain complications and sequels which are due to the orginal disease, and not to the operative treatment: these will always deserve careful consideration.

The main subjects to be considered in this chapter are:

1. The Causes of Death after Operation, including Shock, Peritonitis, and Pneumonia.
2. The Vicious Circle after Gastro-jejunostomy.
3. Peptic Ulcer of the Jejunum after Gastro-jejunostomy.
4. Internal Hernia.
5. Gastric Fistulæ.
6. Duodenal Fistulæ.
7. Subphrenic Abscess.

1. **The Causes of Death after Operation.**—The chief of these are Peritonitis, Pneumonia, Shock, and the Vicious Circle. Unfortunately most writers, while narrating their successes in glowing terms, have not dwelt particularly on the details of their fatal cases, giving only general expressions of opinion as to the most frequent cause of death.

In a series of 92 operations for benign diseases of the stomach, done by the senior author in the Lankenau Hospital, the records of which were analyzed by Whiting, there were 8 deaths. In two patients (Nos. 1 and 8), one operated on in 1900, the other in 1903,

the cause of death is not noted in the records. Death was attributed to exhaustion in one patient (No. 47), referred to at page 108, who was almost exsanguinated from repeated hemorrhages before operation, but lived for forty-eight hours afterward. Two patients (Nos. 7 and 27), one in 1902, the other in 1905, died of peritonitis, which was in each instance due to leakage of the Murphy button employed in doing an entero-anastomosis for vicious circle some days after the primary operation (posterior gastro-jejunostomy with a long afferent loop). One patient (No. 20), operated on in 1904, died from the effects of the vicious circle following the posterior "long loop" operation, relief not being obtained from a secondary operation in which the distended afferent loop was resected with end-to-end anastomosis. One patient (No. 82) with phthisis died of edema of the lungs developing on the eighth day after the operation; and one patient (No. 67), with subacute perforation of a gastric ulcer, died three weeks after operation from nephritis and myocardial disease.

In a second series of 597 operations for benign disease of the stomach, there were 29 deaths (4.95 per cent.). Death was attributed to the following causes:

Pneumonia	6
Peritonitis	5
Shock	4
Myocarditis	3
Acute cardiac dilatation	2
Hemorrhage	2
Obstruction	2
Toxemia	2
Asthenia	2
Pulmonary edema	1
	29

The causes of death recorded in other large series of operations for benign affections of the stomach may be seen in the accompanying tables:

Beckman (Mayo Clinic, 1913): 465 operations, 14 deaths (3 per cent.).

Cause of death:

Pulmonary disease	13
Thrombophlebitis	1
	14

Von Eiselsberg (1914): 334 operations, 17 deaths (5 per cent.). Cause of death:

Hemorrhage from ulcer	5
Anemia following hemorrhage	2
Progressive ulcer	1
Vicious circle	2
Heart failure (4 weeks after operation)	3
Peritonitis	1
Pneumonia	1
Paratyphoid fever	1
Enteritis and marasmus	1
	17

Braun (1914): 75 operations, 8 deaths (10.6 per cent.). Cause of death:

Hemorrhage from ulcer	1
Hemorrhage from suture line	2
Suture insufficiency	2
Pneumonia	1
Vicious circle	1
Pyschosis	1
	8

It is, of course, true that in some of these patients the operation itself cannot be held directly responsible for the fatal termination; but nevertheless it is probable that many of the patients would not have died so soon if no operation had been undertaken.

In a series of 189 operations for *carcinoma of the stomach*, by the senior author, there were 35 deaths (18.5 per cent.):

Among 81 gastro-jejunostomies there were 17 deaths (21 per cent.).

Cause of death:

Exhaustion	5
Peritonitis	4 (none in the last 50 operations)
Shock	2
Toxemia	2
Uremia	2
Cardiac disease	2
	17

Among 39 partial gastrectomies, 12 deaths (10 per cent.).
Cause of death:

Exhaustion	2
Peritonitis	3
Shock	3
Pneumonia	2
Acute cardiac dilatation	1
Fecal fistula	1
	12

One case of total gastrectomy, death from shock.
Three cases of transgastric excisions, one death from shock.
Among 39 exploratory operations, 4 deaths (20 per cent.).
Cause of death:

Shock	3
Uremia	1

The causes of death recorded in other large series of operations for carcinoma of the stomach may be seen in the accompanying tables:
Altschul (1913): Anterior Gastro-jejunostomy, 88 operations, 31 deaths (35.2 %).
Cause of death:

Peritonitis	9	(5 from perforation of button used in anastomosis, 1 from suture insufficiency, 3 from operative technique)
Pneumonia	8	
Vicious circle	2	
Obstruction of bowel	1	
Marasmus	3	
Anemia	2	
Cardiac disease	4	
Phthisis	1	
No autopsy	1	

Posterior Gastro-jejunostomy, 95 operations, 18 deaths (18 per cent.).
Cause of death:

Peritonitis	3
Pneumonia	6
Pulmonary embolism	2
Cardiac disease	2
Cachexia	2
Hemorrhage from tumor	1
Vicious circle	1
Gangrene of colon	1
	18

Partial Gastrectomy, 64 operations, 26 deaths (40.6 per cent.).
Cause of death:

Peritonitis	7
Pneumonia	9
Cachexia	4
Cardiac disease	2
Pleuritis	1
Gastric atony	1
Intercurrent dysentery	1
No autopsy	1
	26

Jejunostomy, 5 operations, 4 deaths (80 per cent.).
Cause of death:

Peritonitis	2
Pneumonia	2
	4

Exploratory Laparotomy, 139 operations, 15 deaths, (10.8 per cent.).
Cause of death:

Peritonitis	2	(one from perforation by the cancer).
Pneumonia	6	
Pulmonary embolism	2	
Cerebral embolism	1	
Marasmus	1	
Cardiac disease	1	
Gangrene of colon	1	
No autopsy	1	
	15	

One case of total gastrectomy, death in 2 days.
Weil (1913), 135 partial gastrectomies, 31 deaths (23 per cent.).
Cause of death:

Peritonitis	18
Pulmonary disease	8
Shock	2
Cachexia	1
Cerebral embolism	1
Acute enteritis	1
	31

The subject of the Vicious Circle after gastro-jejunostomy deserves a section to itself. It will be sufficient here to refer rather briefly to

the subjects of shock, pneumonia, and peritonitis, complicating and following operations on the stomach.

Shock is chiefly due to undue prolongation of the operation on the surgeon's part, or to pre-existing cachexia on that of the patient. It is the surgeon's duty, so far as in him lies, to select that form of operation the unavoidable shock of which that patient will be able to withstand. Ability so to select this operation comes to the surgeon as the result of experience, and is with difficulty learned from a text-book. It should, however, be the surgeon's desire always to aim in the direction of safety, remembering that excellent motto "*primum non nocere.*" In the case of very weak patients even an exploration may seem contra-indicated; in others, gastrostomy, jejunostomy, or even gastro-enterostomy can be performed, and a radical operation, when indicated, may be postponed until some strength shall have been gained by forced feeding.

When the operation has once been undertaken, the actual technique of its performance will influence very materially the development of shock. We always lay great stress on maintaining the natural heat of the body. To this end the patient should wear a jacket of cotton wadding, and his lower extremities should be similarly clothed. In addition to these precautions a hot-water bed which covers the entire top of the operating table may be used. We believe the use of an electrically heated mattress is dangerous: it is too easily overheated. These measures joined to the tonic treatment to which the patient has been submitted during the day or so immediately preceding the expected operation, will in the vast majority of cases prevent the occurrence of shock. Indeed, recently we have observed that patients recover as blithely from even extensive gastrectomies as they do from an "interval" operation for appendicitis. When the bodily heat is thus maintained, and when the surgeon eventrates no viscera except those immediately concerned in the operative procedure, the actual duration of the operation seems to have little tendency to produce shock, at least in the case of chronic lesions. Five minutes more consumed in an operation will very rarely be prejudicial to the patient, and will certainly enable the surgeon to do the operation more thoroughly, and therefore with more prospect of ultimate success, than if he is continually trying to establish a record. We regard thirty minutes as a short time to spend in doing a gastro-jejunostomy, and are not at all ashamed of taking more than twice as long in difficult cases of gastric surgery.

Hemorrhage predisposes to shock. The surgeon, however, who

pursues a definite plan in his gastric operations, and has the necessary acquaintance with the anatomy of the parts, is not apt to encounter uncontrollable hemorrhage. Indeed, in all typical operations there should be no bleeding, as every blood-vessel is clamped or tied before it is divided. Yet where the adhesions are very dense, and where the anatomical landmarks are with difficulty distinguishable, rather profuse hemorrhage may be encountered. The senior author has been forced to abandon a gastrectomy for cancer, and resort to gastro-jejunostomy, on account of furious hemorrhage among pyloric adhesions.

Pneumonia.—Among the authors' patients there have been very few deaths from pneumonia: 8 deaths in nearly 800 operations on the stomach. Beckman (1913), from the Mayo clinic, reported 13 deaths from pulmonary disease among 465 operations all for benign disease; Altschul (1913) 31 deaths from pneumonia among 391 operations, all for malignant disease. We attribute the absence of pneumonia as a postoperative complication in our own experience largely to the precautions, already mentioned, which are taken against chilling the patients; but chiefly to the semi-sitting posture assumed as soon as the effects of the anesthetic pass off, and particularly to the use of nitrous oxide and oxygen anesthesia in very debilitated patients.

It is well known that pneumonia is more liable to follow operations in the region of the upper abdomen than those in the pelvis; and this predisposition has been attributed to various causes. The anesthetic could have no more harmful influence in one case than in the other; indeed, according to Krecke, as long ago as 1901 v. Mikulicz reported a much higher mortality from pneumonia after gastric operations under local anesthesia than when a general anesthetic was employed. However, it is certainly well for the anesthetist to bear in mind that the visceral peritoneum is insensitive (Lennander), and that when once the abdomen has been opened, comparatively light anesthsia is required until the time comes for closing the incision in the abdominal wall.

We are inclined to agree with Kelling (1905) who held that post-operative pneumonia in these cases is produced either by inhalation or by direct infection through the diaphragm; its much greater frequency in malignant and infectious conditions, than in cases of simple pyloric stenosis or gastric dilatation is well recognized. Others have taught that the incision in the epigastric region of the abdomen interferes with deep breathing after the operation, and, the patient restricting his respiratory excursions as far as possible on account of pain, in this way the smaller bronchial tubes become clogged with mucus, hypostatic

congestion is induced, and the onset of pneumonia favored. To prevent respiratory difficulty during the operation, we have the patient's arms laid beside him on the table, never pinned up over his chest. The elaborate study of Cutler and Hunt (1920) on post-operative pulmonary complications leads them to adopt the view that they are with very few exceptions of embolic origin; embolism which may occur through the lymphatics or the blood stream, is favored, as they point out, by sepsis, by trauma, and by the mobility of the part, factors which are all present in many operations on the upper abdominal viscera.

Since surgeons have adopted the habit of sitting their patients up in bed soon after the operation, the prevalence of post-operative pneumonia in gastric cases has markedly diminished; and thus this practice, begun with the erroneous idea that the stomach was emptied by gravity, has been productive of good results, in spite of its mistaken purpose.

As urged by Robson, it is well to direct these patients to inhale deeply three or four times every hour or so, in the hope that thus pneumonia will be prevented, by ridding the terminal bronchioles of accumulated secretion.

It has long been our belief that the injudicious use of saline solution intravenously, as a remedy or supposed preventative of shock, had a tendency to predispose to pulmonary complications, and especially to edema of the lungs. Its value in hemorrhage may perhaps be allowed; but when care is taken to prevent the dissipation of bodily heat it is very exceptional indeed in our experience for patients to be so shocked during any operation, unattended by hemorrhage, as to require the use of saline solution intravenously.

Should pneumonia unfortunately develop, no time should be lost in applying dry cups to the patient's chest, and adopting vigorous medical treatment and putting the patients in the open air.

Peritonitis.—Among the senior author's patients up to 1909 there had been 2 deaths from peritonitis in the benign series. In these patients the primary operation had been a posterior gastro-jejunostomy with long loop (the first operation in 1902, the second in 1905); in both a secondary entero-anastomosis was done on account of the development of the *circulus vitiosus;* and in both leakage occurred after the second operation, in which the Murphy button was employed, and death followed some days later from peritonitis. This form of operation (long loop) has not been employed since September, 1905, and the use of the Murphy button is avoided whenever possible. Since 1909,

there have been 5 deaths from peritonitis among 597 operations of all kinds for benign disease; and among a total of 189 operations for malignant disease there have been 7 deaths from peritonitis.

Simplification of technique, and more especially the popularization of rubber-covered clamps in abdominal surgery, has nearly eliminated peritonitis as a post-operative cause of death in benign diseases of the stomach. Occasionally, to be sure, a death after operation is encountered from the subsequent perforation of a gastric or duodenal ulcer; but these are very rare cases, as are also the cases in which peptic ulcer of the jejunum develops and perforates before convalescence is established.

But it cannot be denied that peritonitis is much more to be feared in malignant than in non-malignant disease, and there probably always will be a small proportion of deaths due to unavoidable peritonitis. In the last 50 gastro-jejunostomies for carcinoma (up to 1920) there had been no deaths from peritonitis, but there were 4 deaths among the preceding 31 operations of gastro-jejunostomy. In the first of these cases (Feb. 3, 1905), the operation was a posterior gastro-jejunostomy with long loop, with primary entero-anastomosis by a Murphy button; the patient died on the sixth day, and at autopsy an abscess was found between the loops of small intestine concerned in the entero-anastomosis, thus making the third fatal case to be attributed to the use of the Murphy button. In the second case (Dec. 14, 1905), posterior short loop gastro-jejunostomy was done, and the patient died on the eleventh day from perforation of a jejunal ulcer on the proximal side of the gastro-enterostomy. In the third case (March 12, 1906) the fatal result is to be attributed to the diseased condition of the gastric wall at the site of the anastomosis. This we think is the element that can never wholly be eliminated as a cause of peritonitis in these cases. In the patients treated by partial gastrectomy it usually will be possible to cut so wide of the diseased area that the sutures will hold securely; but where only a palliative operation is attempted, the surgeon, rather than do nothing, or rather than do a jejunostomy, will often be tempted to make an anastomosis in a portion of the stomach wall already affected, and thus run the risk of peritonitis developing. The fourth and last of the carcinoma patients to die from peritonitis after a palliative operation (April 13, 1907) had a malignant growth involving the pylorus, the gastro-hepatic omentum, the transverse mesocolon, and the pancreas, causing, in addition to the pyloric obstruction, a stenosis of the transverse colon and dilatation of the gall bladder. The operations done were: posterior gastro-jejunostomy, with short

loop; cholecysto-colostomy; and entero-colostomy. Death occurred on the fourth day from fibrino-purulent peritonitis.

Three patients died from peritonitis after partial gastrectomy for carcinoma (among 46 such operations). In two no leakage of the sutured areas could be detected after death; in the third this was the cause of the peritonitis.

II. The Vicious Circle after Gastro-jejunostomy.—By the **Circulus Vitiosus** was formerly understood a train of post-operative symptoms believed to be due to the continued escape of gastric contents by way of the pylorus, and the return of the duodenal contents into the stomach through the afferent loop by way of the gastro-intestinal anastomosis. Fowler (1902) suggested the term *reflux* to indicate that condition where the duodenal secretions (afferent loop) or those of the jejunum (efferent loop) passed into the stomach through the gastro-intestinal anastomosis and produced vomiting, but where, for one cause or another, the gastric contents did not obtain access to the afferent loop through the pylorus. Surgeons have thus spoken of the *duodenal* and the *jejunal reflux*, or have designated the latter condition as intestinal regurgitation. In general, however, the expression *vicious circle* has been indiscriminately applied to pernicious or persistent vomiting after gastro-jejunostomy; and as our knowledge of the normal physiology of the stomach has increased, and as our ideas of the mechanism of the operation of gastro-jejunostomy have been very materially altered within recent years, it is scarcely desirable, even were it possible, to make a distinction in the cause, where we can perceive no difference in the result. Indeed, it is extremely probable that in most cases of gastro-jejunostomy by lateral anastomosis, with open pylorus, precisely the course of events occurs which is stated above to have been the supposed cause of the vicious circle.

A discussion of the supposed *causes* of this condition is, however, of historical interest. No less a surgeon than Terrier has tersely said that it is due to faulty operating—in other words, that it is avoidable by proper technique. Certain it is, that with increased experience the surgeon encounters it less often, and that long series of operations have been reported by various surgeons, without having it once occur. With the modern operation of gastro-jejunostomy it is rarely if ever seen; and although most surgeons have abandoned the anterior operation (save in certain cases of carcinoma) as well as posterior operations with a long afferent loop, because they believe these more apt to be followed by the vicious circle than the "no loop" method originated by Petersen and popularized among surgeons of Great Britain and America

by Moynihan, yet there are still a few surgeons, including Paterson of London, who persist in employing anterior gastro-jejunostomy and who obtain entirely satisfactory results.

Ever since the operation of gastro-jejunostomy was first done, without premediatation, by Wölfler, in 1881, surgeons have been seeking some method by which this vicious circle could be avoided. It is needless here to describe all the technical changes which the operation has undergone in attempting to eliminate this complication. Each individual method has been adopted to overcome what the surgeon believed was the cause of the vomiting. Those who thought it was due to spur formation at the site of the gastro-jejunostomy wound, aimed to prevent this by attaching the jejunum to the stomach for some distance both above and below the opening (Hadra, 1891; Lauenstein, 1896); those who thought it was due to contraction of the anastomotic opening took measures to insure its patency (excision of mucosa, Littlewood; Moynihan); those who thought it was produced by pyloric regurgitation, obliterated the pylorus (Mayo, 1903); and those who thought that it depended on the discharge of the contents of the afferent loop into the stomach, or on obstruction to the discharge from the afferent into the efferent loop, took measures to overcome this difficulty—some doing an entero-anastomosis between the afferent loops (Lauenstein, 1890; Braun; Jaboulay), and others still further complicating the operation by constricting (Wölfler; Chaput; v. Hacker; Fowler) or actually dividing (Doyen) the afferent loop between the entero-anastomosis and the gastro-jejunostomy. But the fact remained that no one surgeon was able to assign a satisfactory cause for the condition, nor always able to avoid it however great his experience may have been with the operation, or with this much dreaded sequel. The theories of Chluniskij (spur formation), of Steudel (contraction of the opening in the transverse mesocolon) and of Kelling, have all been disproved in some instances; and although we are forced to the rather

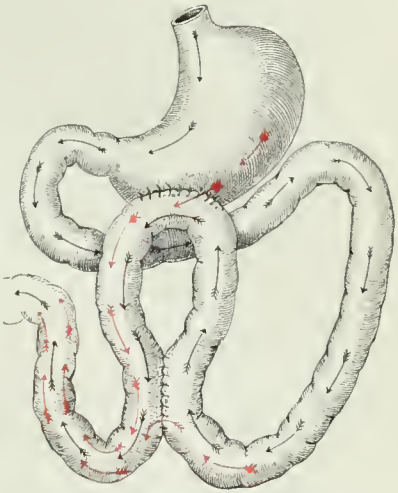


FIG. 127.—Diagram to Show Course of Gastric and Duodenal Contents. Black Arrows Indicate Normal Course, Red Arrows Indicate Course after Anterior Gastro-jejunostomy with Entero-anastomosis.

humiliating conclusion that we do not know definitely what the actual cause is, we are at any rate convinced that it is best avoided by adopting a simplified instead of a complicated technique, and by giving occasion for the production of as few post-operative adhesions as possible.

The theories suggested to explain the vicious circle may be classed under the following heads:

1. The presence of **bile or pancreatic juice in the stomach.** Although excessive amounts of these may cause nausea, acid eructations, and even annoying vomiting, they cannot alone be held sufficient to account for severe cases of the vicious circle. Dastre's experiments on dogs, and operations of cholecyto-gastrostomy by Terrier and others, show that bile is well tolerated by the stomach; while the success of Moynihan's well-known case, in which, for rupture of the intestine at the duodeno-jejunal juncture, the proximal end of the bowel was closed and the distal united with the stomach (see p. 308), thus forcing all the duodenal contents to traverse the stomach before reaching the jejunum, effectually sets aside the theory that pancreatic juice in the stomach is solely responsible for the symptoms. Indeed, as already remarked, it is extremely probable, if not absolutely certain, in most of the gastro-jejunostomies done by lateral anastomosis at the present day, that the secretions of the afferent loop mix freely in the stomach with the gastric contents, and really are of benefit to the patient (p. 111).

2. The location of the anastomotic orifice at some other than the **"most dependent portion"** of the stomach. This is assuredly not a cause, in itself, since we now know, and might have known twenty years ago, if we had heeded the researches of Kelling, that the stomach empties itself only by contraction, not by gravity. Moreover, many successful operations have been done without regard to the location of the anastomosis at the "most dependent point." Indeed, that point where the anastomosis is made may soon become the "most dependent point" by the drag of the intestine; and yet what is the most dependent point when the patient is erect, will not be so when he is in bed. All of which shows the folly which inspired surgeons who regarded the stomach as a tin can which to be drained must have a hole cut in its bottom, and must then be kept on end if it were to be kept empty.

3. The presence of a **long afferent loop** has been blamed, but, as already mentioned, many entirely successful operations disprove this theory, and we must look elsewhere for a cause.

4. **Obstruction at the gastro-jejunal anastomosis,** of some form or other, is, we believe, the true cause of this condition. The vicious circle was more frequent in the earlier operations because the surgeon

damaged the bowels and the stomach more, and produced temporary paresis, or more lasting adhesions; or because the operation was followed by the formation of a spur, a kink, a valve of mucous membrane, or some other form of mechanical obstruction. Kelling very clearly pointed out that if the stomach was damaged (either by the operation or by previous disease, as in far advanced cancer cases), it could not properly contract after the gastro-jejunostomy; and that under such circumstances the intestines would empty themselves into it. He insisted, moreover, on the gastro-duodenal reflex, by virtue of which the gastric contractions cease so long as the duodenum remains full. If, therefore, there was a patulous pylorus, or if with obstructed pylorus the gastric contents by reverse peristalsis gained access to the duodenum (the afferent loop), the stomach could no longer empty itself by peristalsis until the duodenum was emptied; and if this was prevented by an obstruction at the site of the anastomosis, circumstances were very favorable for the the development of the vicious circle. We will recur again to this topic when discussing the symptoms of the vicious circle.

The employment, then, of a suitable technique will prevent obstruction to the afferent loop at the gastro-jejunal anastomosis, and will prove the correctness of Terrier's contention that the vicious circle is due to faulty operating.

The **symptoms** of the vicious circle usually do not develop for several days after the operation. At first there may be merely a slight regurgitation of bile-stained fluid; later, when more food is taken, upper jejunal contents may be vomited, the regurgitation then taking place from the efferent loop.

Several cases have come under our observation in which convalescence after the gastro-jejunostomy was satisfactory, but occasionally there would be copious vomiting of biliary matter. Meals were eaten with appetite, no discomfort ensued, but three or four hours after the meal this copious biliary vomiting would occur. The patients did not lose in weight. One patient gained forty pounds during the first year after the gastro-jejunostomy, but the vomiting of pancreatic and bilious fluids was so persistent and annoying that she finally submitted to another operation.

There were sometimes seen instances of the vicious circle which more imperatively demanded relief than those patients just mentioned. In such cases the vomiting was persistent from the time of the operation, emaciation was rapid, and unless something had been done speedily to relieve the patients, they would have died of exhaustion

and inanition; in fact, all the symptoms of a high intestinal obstruction were present.

The **treatment** should at first be palliative. The patient should be sat up in bed, the stomach washed out, and all food by the mouth stopped. Enteroclysis should be employed if mouth feeding cannot be resumed promptly. When vomiting has been absent for twenty-four or thirty-six hours, a very little water may be given by mouth—a teaspoonful every hour or two; but this must again be stopped if the vomiting reappears. In the less severe cases it is sometimes sufficient to employ lavage every second or third day; light diet, in small quantities, being taken meantime. We have known a patient content to live in this way for some months.

If a cure is not spontaneously effected thus, or immediately if the symptoms are urgent, the abdomen should be reopened, and mechanical correction of the obstruction attempted. Operation should not be postponed so long that the patient's strength will not be sufficient to stand the shock; and, on the other hand, too extensive an operation should not be employed on such debilitated patients.

The following case, which has been published elsewhere in detail by the senior author, is quoted here as illustrating many points of importance in this connection.

A young woman of 24 years, who for three years had presented symptoms of gradually increasing pyloric obstruction, was operated on at the German Hospital, by Dr. John B. Deaver, July 8, 1903. Numerous adhesions were found about the neck of the gall-bladder and duodenum. The gall-bladder was normal, but slightly distended; the stomach was slightly enlarged, somewhat proptosed, and the pylorus was thickened. A posterior trans-mesocolic gastro-jejunostomy was done, with sutures, the afferent loop being about 25 cm. (ten inches) long.

The patient did well and was free from vomiting for five days. On July 13th, 700 cc. of dark green bile were vomited; her general condition was good.

July 15th. Patient vomited bile during the night. Appetite good, feels better than she has in years. Stitches removed. Wound healed.

July 17th. Patient vomited bile with small portion of fecal matter.

July 18th. During early morning the patient vomited; vomitus stercoraceous in character, about 300 cc.; and an intestinal obstruction was believed to have taken place necessitating a second operation.

Second operation, July 18, 1903 (ten days after the first operation). Omentum found adherent to abdominal scar; adhesions separated, and omentum and transverse colon were turned upward. This reflection upward carried several coils of small bowel along, and it was found that the proximal and distal limbs of the anastomosed loop were firmly adherent to the posterior layer of the transverse mesocolon, interfering to a marked degree with the peristalsis of the bowel. The adhesions were all separated, and the denuded surfaces were inverted with silk sutures, or covered with Cargile membrane. The gastro-jejunostomy was apparently perfect. An entero-enterostomy was then performed, by sutures, 15 cm. (six inches) from the gastric anastomosis. (See Fig. 127.)

There was some vomiting of green material upon the two days following operation but this stopped upon the use of lavage.

July 25th. Stitches removed, wound healed.

July 26th. Patient vomited twice about 300 cc. of light greenish material. Wine of ipecac, 10 drops every hour, was given.

July 27th. No vomiting.

July 31st. Ipecac stopped.

Aug. 2nd. Vomited 300 cc. of light green material about 10 p. m. Vomiting could not be attributed to anything eaten. Given 10 drops of wine of ipecac every two hours during day.

Aug. 7th. Vomited small quantity of yellow material. Ipecac stopped.

Aug. 12th. Discharged. Condition fine; is gaining weight, and has not vomited since Aug. 7th.

On Sept. 14, 1903, this patient was readmitted to the German Hospital. She stated that on Aug. 16th, four days after her discharge, vomiting had recommenced, at first of bile and later of food and bile. When admitted she vomited everything given by mouth, and large quantities of thick, ropy, dark green material, with a very strong odor. Feeding by rectum was begun, but the patient continued to vomit the thick, dark green material, containing large quantities of bile. On washing out the stomach large quantities of the same material were obtained. She was well nourished, notwithstanding the vomiting, proving that the food must have been digested in great part, especially as the patient did not seem to have lost much weight. Examination of the eyes was negative; the pelvic organs were normal; and no constitutional cause for the vomiting could be found.

Third Operation, Sept. 19, 1903. The omentum was found universally adherent, and there were dense adhesions between the coils of bowel. The gastro-jejunostomy was exposed, and the opening found to be freely patent. The entero-enterostomy was exposed, some difficulty being encountered in distinguishing the anastomosis by reason of the numerous adhesions. When these were seperated the anastomosis was found to be in perfect condition, with some sacculation. Adhesions were further separated throughout the abdominal cavity. The entire omentum was ligated and cut away. The gall-bladder was found normal in size, numerous adhesions surrounding it; the stomach was normal in size. By means of a pedicle needle a piece of silver wire was passed around the pylorus and tied. The abdominal cavity was filled with normal salt solution, and the abdominal wound closed by tier suture. The patient was shocked, the pulse being barely perceptible at the close of the operation. An intravenous injection of saline solution was given before she left the operating room.

Sept. 20. Patient vomited bile several times during the day.

Sept. 22. Buttermilk ordered. Patient feels somewhat distressed in the epigastric region, no vomiting.

Sept. 23. Patient feels well; no epigastric distress.

Sept. 29. Eating light diet. No nausea or distress.

Oct. 10. Patient vomited after breakfast.

Oct. 11–15. Patient vomited several times each day; complained of some abdominal pain; there was some tympanites. For symptoms of obstruction, it was determined the next day to re-open the abdomen.

Fourth Operation, Oct. 16, 1903. A coil of small intestine was found closely adherent to the parietal peritoneum. The intestines were found universally adherent to each other, and to the remains of the omentum, binding together the transverse, ascending and descending colon and sigmoid flexure, and various loops of small intestine, one to another. No portion of the bowel, excepting about five feet of the ileum, was free from adhesions. The adhesions were separated, bleeding points ligated, and all denuded surfaces covered with Cargile membrane. The entero-enterostomy and the gastro-jejunostomy were examined and found patulous. At no portion were the intestines collapsed or

unduly distended. The abdomen was closed by through-and-through sutures of silkworm gut.

Oct. 17. Much vomiting.

Oct. 19. No vomiting.

Oct. 29. Sutures removed; wound healed.

Nov. 2. Patient allowed to sit up in a chair. No vomiting; feels strong.

Nov. 9. Patient walking about.

Nov. 13. Patient went home.

The patient was at home for ten days when she again began to vomit as before: in the morning bile; later in the day particles of food; would vomit two or three times every day. On re-admission her nutrition was good. On the abdomen were two scars of former operations. Slight distension of the stomach.

Fifth Operation, Dec. 7, 1903. An incision seven inches long was made, dissecting out the last cicatrix. On opening the peritoneum many adhesions were found between intestines and under surface of incision. Universal adhesions were present throughout the intestinal canal; these were carefully dissected free, and two holes in the intestine, which were accidentally made, were closed with sutures of silk. Abdomen was filled with salt solution, and wound closed with through-and-through sutures of silkworm gut.

Jan. 14, 1904. Patient apparently entirely well. She remained in good health for one year, and then died of unknown cause, but with no gastric symptoms.

This unfortunate woman, therefore, had undergone five operations, one after the other, for the relief of severe vomiting. An entero-anastomosis and an occlusion of the pylorus both had failed to relieve her condition. Every cause for vicious circle or for jejunal reflux seemed to have been eliminated, except the influence of adhesions, which were encountered at each operation. The patient gained in weight even while vomiting, indicating that the digestive power was not seriously impaired.

The senior author's experience with the vicious circle (up to 1913) embraced twelve cases, which may be thus classified:

1. Among 25 operations of posterior gastro-jejunostomy, with long afferent loop, and without primary entero-anastomosis.	Eight developed the vicious circle; 5 of these recovered, and 3 died, after secondary operations.
2. Among 19 operations of posterior gastro-jejunostomy with long afferent loop, and with primary entero-anastomosis	Three patients (one more than 5 years after operation; another nearly 8 years after operation): two were cured after secondary operation, the third died after the second secondary operation.
3. Among 429 operations of posterior gastro-jejunostomy with "short loop," or with "no loop," (up to Jan. 1, 1920)	One patient, who was cured by a secondary operation. There has been no case of vicious circle since the year 1915.

Probably no more forcible comment is needed on the disadvantages of the long loop posterior gastro-jejunostomy.

The eight patients in the category where no primary entero-anastomosis was done, who developed the vicious circle, were reoperated on at intervals varying from four days to one year after the primary

operation. In six cases an entero-anastomosis was done at the secondary operation; four of these six patients were thus relieved of their symptoms, but the remaining two patients died after the secondary operation from peritonitis due to leakage of the Murphy button employed in making the anastomosis. The seventh patient was treated (April, 1904) by resection with end-to-end anastomosis by suture of the distended afferent loop, thus converting the operation into one of the modern short loop gastro-jejunostomies; but this patient died two days after this secondary operation, of exhaustion. The last of these eight patients was operated on for the vicious circle one year after gastro-jejunostomy by this method (posterior long loop, without entero-anastomosis) had been done in another hospital by another surgeon. In this patient relief was obtained by the performance of an entero-anastomosis, ligation of the pylorus, and ligation of the afferent loop (Fowler). She was last heard from more than two years after this secondary operation, and was in good health, and feeling much better than before this last operation, though still somewhat troubled by gastric symptoms.

The first patient who developed the vicious circle among the series of long loop gastro-jejunostomies in which a primary entero-anastomosis was done, was entirely relieved of his symptoms by a secondary ligation of the pylorus, employed three months after the primary operation. He was last heard from two and a half years after the secondary operation, was in excellent health, and had no symptoms referable to the stomach. The second patient, who developed symptoms of the vicious circle in 1911 (5¼ years after the original operation) recovered after having the duodenum plicated, but has not been traced. The third patient developed symptoms in 1913, nearly 8 years after the original operation; it was found that the afferent and efferent jejunal loops made a spur, which obstructed the gastro-jejunostomy opening, though the entero-anastomosis was patulous and apparently functionating. Release of some adhesions caused this spur to disappear. As the pylorus was patulous the duodenum was plicated just beyond it; and the appendix was removed. For recurrence of symptoms the abdomen was re-opened a week later; the afferent jejunal loop was sectioned, its gastric end was closed, and its duodenal end was implanted into the efferent jejunal loop (Y-anastomosis), but the patient did not rally from the extensive operation.

In the third series of cases (posterior gastro-jejunostomy with a short loop), there was one patient, operated on in December, 1905, who developed regurgitant vomiting. Five months later it was found

at the secondary operation that the gastro-jejunostomy opening was patent; that the pylorus was obstructed, but that the short proximal loop did not appear to be draining well. Finney's pyloroplasty was therefore done, as well as an entero-anastomosis between the short afferent loop and the efferent loop of the jejunum. Recovery was uneventful; but although the regurgitant vomiting was relieved, the patient when last heard from, over two years later, could only be classed among those "much improved" by operation. In looking back at this case it seems not impossible that it was one of those in which, as pointed out by Mayo (1908), the jejunum has attached to it for some distance from its origin a peritoneal fold running from the transverse mesocolon, and that owing to failure to recognize this anomaly, the operation instead of being a short loop gastro-jejunostomy became in reality a long loop operation. Similar cases have been encountered since, but in these the peritoneal fold was recognized, divided, as recommended by Mayo, and the usual operation done, with the usual satisfactory result.

In common with all surgeons who did stomach surgery as long as twenty five or more years ago, the senior author's earliest gastro-jejunostomies (for carcinoma) were done on the anterior wall of the stomach, by means of the Murphy button. It was not long, however, until the posterior operation was adopted, still employing the Murphy button. In the anterior operation a long afferent loop of jejunum is obligatory, on account of the necessity of spanning the transverse colon and great omentum; and, again in company with other surgeons, the senior author pursued the same technique in performing the posterior operation, not appreciating at that time the drawbacks and the positive dangers of the long afferent loop. Believing that the regurgitant vomiting observed in some of these cases was due to obstruction at the anastomotic opening, preventing the proximal (afferent) loop of jejunum from freely emptying itself into the distal (efferent) loop, the technique was then changed so as to include at every primary operation an entero-anastomosis between the afferent and efferent loops, thus making sure that obstruction to the afferent loop could not exist. This method proved for a long time satisfactory, although its performance consumed more time than the simple gastro-jejunostomy alone. To make this additional time as short as possible, a Murphy button was at first employed in making the entero-anastomosis, but when disaster directly traceable to the use of the Murphy button occurred, this method was abandoned, and since that time simple sutures have been employed.

Even while employing the method of posterior gastro-jejunostomy with the long loop and entero-anastomosis, we were, of course, quite well aware of the brilliant results of other surgeons from the "short loop" and the "no-loop" operations; but as long as the technique being employed proved satisfactory, it seemed poor surgery to change that technique merely to keep in fashion. In the course of time, however, it came to pass that a patient on whom this operation had been done, returned with the vicious circle. This patient, as already described, was entirely relieved of his symptoms by ligation of the pylorus. Since that time the short loop or "no loop" operation has been employed, and the vicious circle is now never a sequel of the operation.

We would recommend, therefore, the following course in the operative treatment of the vicious circle, following posterior gastro-jejunostomy with long afferent loop:

1. Entero-enterostomy between the afferent and efferent limbs of the jejunal loop. If this failed to relieve,
2. Ligation of the pylorus should be the next step, while
3. Occlusion of the afferent loop between the entero-anastomosis and the gastro-jejunostomy may be done as a final step.

Should for any reason the performance of entero-enterostomy be peculiarly difficult, probably the next step would be to divide the afferent loop close to the gastro-jejunostomy, suture its gastric end, and implant the proximal coil (afferent loop) into the jejunum at least eight inches below the gastro-jejunostomy. This would supplant the lateral anastomosis by a posterior gastro-jejunostomy in-Y, according to the method of Roux. Kausch (1903) adopted the plan of anastomosing the efferent jejunal loop with the duodenum in its retroperitoneal portion but the patient died from pulmonary complications. Noetzel (1912) however reported a case in which this method was successful in relieving a vicious circle.

III. Gastro-jejunal and Jejunal Ulcer.—It is probable that ulcers occurring in the jejunum near the gastro-intestinal anastomosis are closely related etiologically to those which develop at the gastro-enterostomy site itself, and which often are spoken of as "marginal ulcers." We can see no very good reason for separating these two types of ulcer pathogenetically, though, as will be seen below, their symptomatology is usually distinct. Attention was formerly centered on the jejunal ulcers, since these called for active intervention because of perforation into the peritoneal cavity; and probably because

they were formerly relatively more frequent than the marginal ulcers, since many more anterior than posterior gastro-jejunostomies were done in the early period of gastric surgery, and it is a well ascertained fact that jejunal ulcer is rarer than gastrojejunal ulcer when the posterior no-loop operation is employed.

According to Gosset (1906), it was not until 1899 (seventeen years after the operation of gastro-jejunostomy was first performed), that Braun reported the first case of peptic jejunal ulcer, which in his patient developed one year after gastro-jejunostomy. The first French case was reported by Quénu in 1902, and the first in England by Mayo Robson in 1904. Hamann (1907) appears to have been the first in America. Moynihan (1908) collected 60 cases of jejunal ulcer following gastrojejunostomy. But the recognition of gastrojejunal ulcers (the so-called marginal ulcer) is due largely to the writings of Paterson (1909), W. J. Mayo (1910) and Carman and Balfour (1915).

We quoted figures in the first edition of this work (1909) showing that jejunal ulcer occurred in less than one per cent. of gastro-jejunostomies, without regard to the special technique adopted; and another set of statistics, of more recent operations, numbering 3869, shows that only in 29 cases did a jejunal or gastrojejunal ulcer develop or about in 0.75 per cent.

The whole subject was reviewed in 1915 by Lieblein, who, after excluding all doubtful cases, admitted 129 which he regarded as certain, 79 of which were jejunal ulcers, and 50 gastro-jejunal. Wright (1919) admits 142 proved cases to his tables (75 or 52 per cent. were jejunal, 63 or 44.3 per cent. were gastrojejunal, and in 4 the ulcer's location was not stated). As to the influence of the type of the preceding operation on the development of this complication, Lieblein quotes Von Roojen's figures. This author collected 189 anterior gastro-jejunostomies with 6 gastrojejunal or jejunal ulcers; while among 444 posterior gastro-jejunostomies he found only 4 cases of jejunal ulcer developed.

Jejunal Ulcer was formerly attributed to the action of the hyperacid gastric secretions on a portion of the intestinal canal insufficiently protected by the alkaline secretions which exist in the duodenum; and the greater frequency of such ulcers after anterior gastro-jejunostomy and after posterior long loop operations in which no entero-anastomosis was done, certainly seemed to support this theory. Robson observed one case of jejunal ulcer among 30 anterior gastro-jejunostomies, but he did not have it occur at all among 300 modern posterior operations. Moreover, this complication had been noted (Connell, 1908) only once (Lennander) after gastro-jejunostomy for cancer, in which disease

gastric acidity is absent or much diminished. Another case following gastrectomy for carcinoma has been recorded by V. Pauchet (1920). But it must be acknowledged that most patients with cancer probably do not live long enough for this complication to be noted. And we think surgeons, and perhaps pathologists also, do not sufficiently realize that peptic ulcer, whether in the stomach, the duodenum, or the jejunum, is possibly as much a symptom of disease as a disease itself; in the same way that the ulcerated Peyerian patches of typhoid fever do not themselves constitute the whole of the disease. Thus it is not beyond the bounds of possibility for future experience to show that peptic ulcers of the jejunum may exist in immature state (hemorrhagic ecchymosis, exulceratio simplex, etc.), in some patients with gastric ulcer, even at the time of operation; and that therefore the subsequent development of perforating or hemorrhagic ulcers may occur in spite of, and not in consequence of, the gastro-jejunostomy.[1] As Lieblein points out, there is no actual proof that gastric acidity causes the ulcer; and more than one ulcer (as many as four or five) may exist.

Symptoms may arise within a few days or not for many years. The shortest interval was ten days; while several cases have not developed until 7 years after the primary operation. Perforation is frequently the first symptom; but in other cases subacute or chronic perforation occurs, and the patients return complaining of a recurrence of gastric symptoms.

Of the 79 jejunal ulcers studied by Leiblein, *perforation* into the free peritoneal cavity occurred in 24 cases (30 per cent.), while the remainder (55 cases, 70 per cent.) ran a chronic course. In 13 cases an internal fistula developed: 10 jejuno-colic fistulæ, 1 gastro-colic fistula, and 2 jejuno-gastro-colic fistulæ. Only 6 patients survived among the 24 cases of acute perforation; all 6 who recovered were operated on, as well as 3 patients who died, a mortality of 33 per cent. for those treated by operation, compared with a mortality of 100 per cent. among those in whom no operation was done. Among the *chronic cases* there were many operations, and all but 18 patients had to have more than one operation (several had 3 or 4 operations), and some had recurrence of symptoms as long as 5 years after the operation for jejunal ulcer. Thus

[1] Blanc and Mossé (1908) narrated the history of a patient with symptoms resembling pyloric obstruction which were found at operation to be due to stenosis of the upper jejunum from ulceration; and Schwarz (1914) observed a stenosing jejunal ulcer in a boy 10 years of age, on whom he was doing gastro-jejunostomy for pyloric obstruction. Ulcers of the jejunum not sequels of gastro-jejunostomy were well studied in 1913 by Cade, Rouhier, and Martin and by Leotta in 1919, who collected 22 such cases involving the jejunum or ileum.

it is evident that treatment of jejunal ulcer has not been very satisfactory on the whole. The simplest of the radical operations for these chronic cases, according to Lieblein (suture of a perforation discovered on separating adhesions) had no mortality, and the end results though not brillant were better than after such palliative operations as establishing a jejunal fistula or making a new gastro-jejunostomy. The ulcer was resected in 15 cases, with 3 deaths.

Gastro-jejunal Ulcer.—Paterson (1909), Mayo (1910) and others have blamed this complication on faulty operating defects in technique); but to us it does not seem just to lay the blame for all gastro-jejunal ulcers solely on the operator: the stomach is diseased, and whatever be the real cause of gastric ulcer, it probably is not removed by gastro-jejunostomy alone; so that development of an ulcer at the site of anastomosis or elsewhere may in many cases be properly attributed, we believe, to the underlying disease. However, this view does not absolve the surgeon from performing the anastomosis with the utmost attention to the details of suture; and it is above all things important to impress on the patient the fact that operation is only one step in the cure of gastric ulcer—that, as has often been pointed out, operation is often only a mechanical device to allow medical, dietectic and other chemical measures to become effective. If this fact were constantly borne in mind, indiscretions and negligences in diet and in oral hygiene would be less apt to occur, and the possibility of jejunal or gastro-jejunal ulcer developing would be correspondingly diminished.

The influence of non-absorbable suture material, used as a through-and-through suture, in causing ulceration, was noted by Key (Berg's case) in 1907, and in 1910 by Mayo; but altogether too much stress has been laid on this lapse in technique, for as such we regard it—only absorbable suture material being suitable in our opinion for through-and-through intestinal sutures. In the majority of cases of gastro-jejunal ulcer which have come under our own observation there was no evidence of an unabsorbed suture trying to ulcerate its way into the lumen of the bowel; and in Woolsey's case (1917), explored two years after primary operation, though a non-absorbable suture hung free in the anastomosis there was no ulcer.

The presence of an ulcer at the site of anastomosis can rarely be certainly diagnosticated, though roentgen-ray studies, as pointed out by Carman and Balfour (1915) may show irregularity at the site of anastomosis or stricture of the jejunum just beyond it. The patients return complaining usually of a recurrence of the symptoms for the relief of which the original operation was undertaken. Of the 50 cases

studied by Lieblein (1915), perforation into the free peritoneal cavity occurred only in 6 (12 per cent.), compared with 30 per cent. of acute perforations among the cases of jejunal ulcer. The free interval following the gastro-jejunostomy was from 4 weeks to 7 years. Five patients with acute perforation of gastro-jejunal ulcers were operated on with 4 recoveries; the only patient who was not operated on, died. In 44 cases the course of the gastro-jejunal ulcer was chronic, and in 21 of these cases a tumor mass formed, palpable either before operation (to the left of the umbilicus or in the left hypochondrium) or after opening the abdomen. The free interval varied from a few months to 8 years. In 10 of these 44 cases obstruction was the predominating feature; and in 4 cases a gastro-colic fistula formed.

Wright's (1919) study indicated that 19 patients died without re-operation; 19 died after the re-operation; 25 were not improved by re-operation; and 82 were relieved by re-operation.

The operation for the relief of gastro-jejunal ulcer may prove very difficult, especially when the ulcer follows the posterior operation, when the parts are buried in adhesions, and subacute or chronic perforations may be uncovered when least expected. Figure 128, after a sketch and anatomical preparation by the junior author, from a patient under his care in the Episcopal Hospital, well shows what may be encountered. Usually the best course to pursue, is to resect the entire anastomosis, and establish a new gastro-jejunostomy as nearly as may be at the same site. In cases of posterior no-loop gastro-jejunostomy, however, this may require removal of so much of the proximal jejunum as to necessitate anastomosis of a lower coil of jejunum to the stomach with termino-lateral implantation of the afferent into the efferent loop; or even Kausch's method (p. 389) of duodeno-jejunostomy may have to be employed to secure drainage of the duodenum if excision and suture of the proximal jejunum causes marked stenosis of the latter. Pauchet (1920), who has operated on 10 patients, says the best operation, if the patient is strong enough, is to resect the jejunum, reestablishing its lumen by end-to-end anastomosis; then do partial gastrectomy, including the site of the former anastomosis in the portion of stomach removed; and, finally to implant the stomach (as in Polya's technique), into the jejunum about 10 cm. below where the latter was resected.

Moynihan (1920) reports 27 operations for jejunal or gastro-jejunal ulcer, with 2 deaths (7.4 per cent.); the Mayo Clinic (St. Mary's Hospital Reports, 1918–1919) records 34 recent operations for these lesions, with only 1 death.

The senior author has done 6 operations (on four patients) for

gastro-jejunal ulcer, as well as one operation for an ulcer developing in the anastomosis of a gastro-gastrostomy for hour-glass stomach 9 years after the primary operation. A summary of these cases is appended:

CASE 1. In 1915 posterior gastro-jejunostomy by another surgeon for duodenal ulcer.

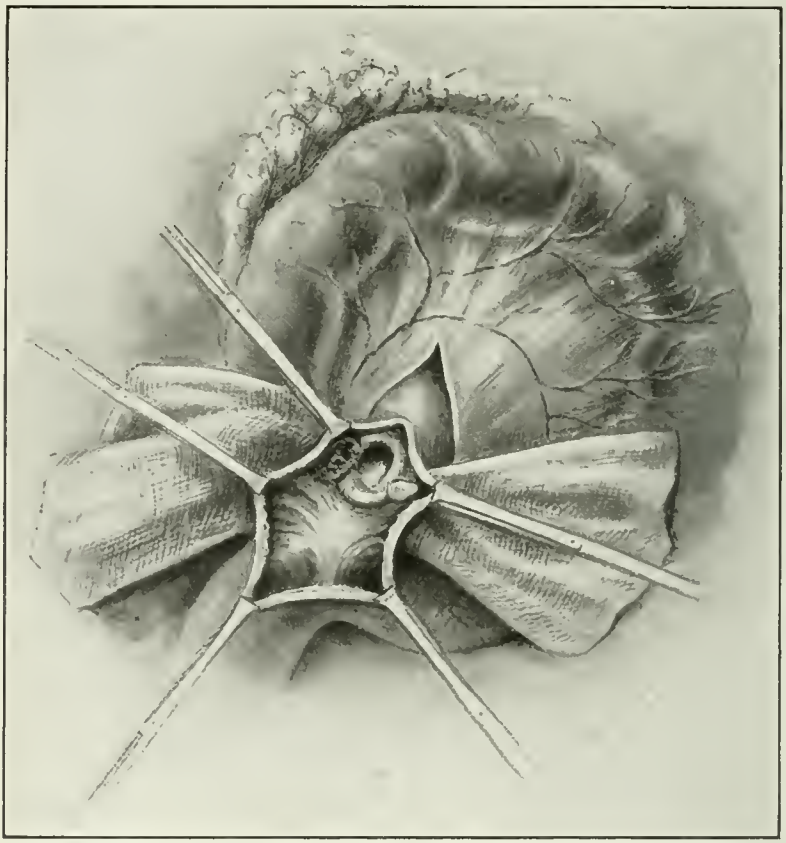


FIG. 128.—Gastro-jejunal Ulcer Following Posterior "No-loop" Gastrojejunostomy Done Previously by Another Surgeon. Gauze has been Passed Beneath the Anastomosis the Interior of which is Exposed by an Incision in the Jejunum (Efferent Loop). Note the *Chronic Ulcer* at the Left of the Anastomosis, and the *Chronic Perforation* (Gauze Showing Through) at the Right. Treated by Resection and a New Gastrojejunostomy. Recovery; but Death Followed a Subsequent Operation (Presumably for Recurrence) Two Years Later, after the Patient's Return to his Home in Austria. From a Patient Under the Junior Author's Care in the Episcopal Hospital.

Oct. 16, 1917, excision of anastomosis and Roux Y-gastro-jejunostomy at Lankenau Hospital for gastro-jejunal ulcer.

April 24, 1919. Anterior gastro-jejunostomy at Lankenau Hospital for recurrence of gastro-jejunal ulcer.

Patient cannot be traced.

Case 2. In 1914 posterior gastro-jejunostomy by another surgeon for gastric ulcer.

Fig. 129.—Specimen Secured by Partial Gastrectomy in a Case in which a Marginal Ulcer Developed 9 Years after Gastro-gastrostomy for Hour-glass Stomach. The Anterior Wall of the Stomach has been Opened.

Aug. 15, 1917, subtotal gastrectomy and Roux Y-gastro-jejunostomy at Lankenau Hospital for gastro-jejunal ulcer.

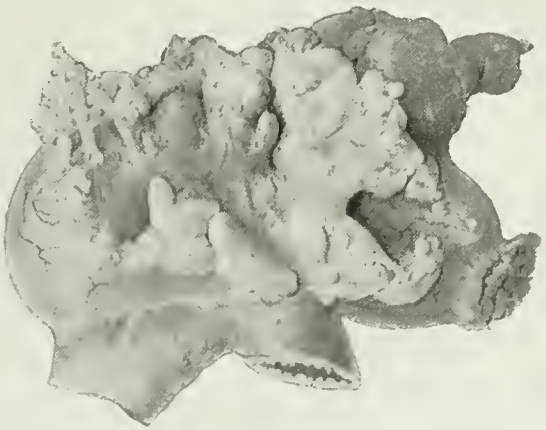


Fig. 130.—Posterior View of the Specimen Shown in Fig. 129. From a Patient Under the Senior Author's Care in the Lankenau Hospital.

Jan. 28, 1919, suture of acute perforation of gastro-jejunal ulcer at Lankneau Hospital.

Jan. 5, 1920 reports in good health, no local symptoms.

Case 3. Nov. 19, 1913, posterior gastro-jejunostomy by another surgeon for perforation of duodenal ulcer.

In 1915, unknown operation on stomach by another surgeon for "peptic ulcer."

March 3, 1916, subtotal gastrectomy, posterior gastro-jejunostomy and entero-enterostomy at Lankenau Hospital for gastro-jejunal ulcer.

Dec. 15, 1917, reports in excellent health.

Case 4. April 8, 1915, posterior gastro-jejunostomy and plication of duodenum for ulcer, at Lankenau Hospital.

Feb. 8, 1917, resection of gastro-jejunal ulcer, and a new posterior gastro-jejunostomy established at Lankenau Hospital.

Jan. 1, 1918, reports in excellent health.

Case 5. March 26, 1907, gastro-gastrostomy at Lankenau Hospital for hour-glass stomach (incidental cholescystostomy).

April 13, 1916, subtotal gastrectomy, posterior gastro-jejunostomy and entero-enterostomy for ulcer at anastomosis. Figs. 129 and 130 represent the specimen secured by partial gastrectomy.

Died 3 months after operation from pulmonary tuberculosis.

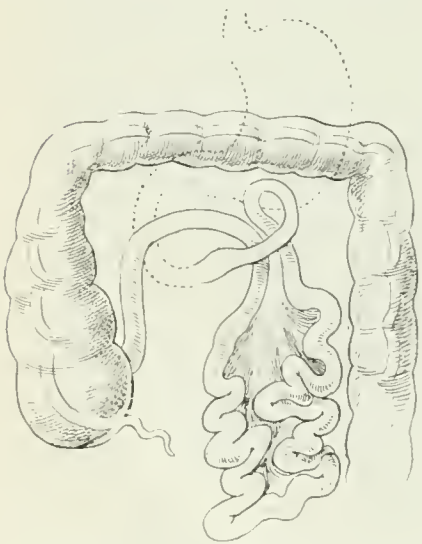


Fig. 131.—Internal Hernia after Gastro-jejunostomy. (*Moschcowitz's Case.*)

IV. Internal Hernia after Gastro-jejunostomy.—This complication is rarely observed. (*a*) *Hernia between the afferent loop and the mesocolon.* Moschcowitz and Wilensky (1915) collected 8 cases of this nature, including one of their own (Fig. 131); and Bryan (1920) adds a ninth case while the case mentioned in the first edition of this work as seen by the junior author in 1902 (in Harte's service at the Episcopal Hospital) makes the tenth case. In 4 of these instances the complication occurred after a posterior short-loop or no-loop operation; in 2 after anterior gastro-jejunostomy; in 2 after a posterior long-loop operation; and in 1 case (Gorden, 1905) the nature of the gastro-jejunostomy is not described. The bowel may enter the jejuno-mesocolic space either from the right or the left. Moschcowitz and Wilensky advise that the afferent loop should always be sutured

to the apposed under layer of the transverse meso-colon, at the original operation.

Even without an actual hernia, the long loop has been responsible for death in a small number of cases by producing obstruction through volvulus of the anastomosed loop, or by drawing the mesentery so taut as to strangulate the lower bowel beneath it.

(*b*) Another sequel, which has followed transmesocolic operations, is the occurrence of a hernia through the opening in the trasnverse mesocolon into the lesser peritoneal cavity. Hartmann, Moynihan, and Harte have each had such a case; but since the possibility of such an event has been recognized its occurrence has been prevented by the nearly universal custom of suturing the edges of the mesocolic opening around the gastro-intestinal anastomosis.

V. Gastric Fistulæ.—These are classified by systematic writers as the Internal and the External varieties.

External Gastric Fistula (Gastro-cutaneous Fistula).—Lieblein and Hilgenreiner in 1905 found more than 120 cases of this unusual affection recorded and Hilgenreiner (1913) subsequently collected 40 more cases. This sequel may follow either lesions of the stomach itself, or affections of neighboring organs. Among the causes are:

1. *Traumatisms.*—Stab or gunshot wounds (as in Beaumont's patient, Alexis St. Martin) were more apt to be followed by the development of a gastro-cutaneous fistula in former times when patients were left either to die of themselves, or to recover in such condition as might be determined by the natural course of events. At present, when practically all such cases are subjected to immediate operation, this sequel is very rare. The stomach may also be perforated from within by foreign bodies which have been swallowed. Lieblein and Hilgenreiner refer to two such cases.

2. *Gastric ulcer* is one of the recognized causes of this condition. It may result from perforation when the stomach is adherent to the abdominal parietes, or secondarily through the external rupture of a perigastric abscess.

3. *Carcinoma of the stomach* was noted as the cause in 26 cases among those collected by Lieblein and Hilgenreiner. As with ulcer, cancer may directly implicate the abdominal wall, or a perigastric abscess may form first. It is also possible for secondary growths in the skin, especially at the umbilicus, to perforate the stomach.

4. *Strangulated hernia* is a very much less frequent cause of gastric than it is of intestinal (fecal) fistula.

Among diseases of neighboring structures, which may cause fis-

tulous tracts to form between the stomach and the integument, must be mentioned, besides inflammatory and malignant diseases of the abdominal wall, affections such as hepatic and subphrenic abscesses; caries of the ribs or sternum; hydatid cysts; cysts of the pancreas, etc.

Prophylaxis is better than cure. In very many cases the only **treatment** that can be attempted will be palliative; moreover, these fistulæ not infrequently heal of themselves. If due to benign disease (perigastric abscess, injury, etc.), an operation may properly be undertaken. Billroth, in 1877, was the first to perform gastrorrhaphy for this condition. By opening the peritoneal cavity to one side of the fistula, and carefully excluding all surrounding organs by gauze packs, it frequently will be possible to dissect the fistulous tract free, and suture the gastric opening. In other cases it may be better to attempt the closure of the fistula by freshening its edges, and transplanting a flap. Patients who cannot be properly nourished, on account of persistent leakage, may be treated by jejunostomy. Patel's patient (1909) survived 8 months with the jejunostomy.

Internal Gastric Fistula.—As the result of disease or injury a fistula may form between the stomach and almost any neighboring organ, or even with another part of the stomach itself. The colon is most often involved (gastro-colic fistula); but communications have been recorded with the duodenum, esophagus, small intestine, gall-bladder; and fistulous tracts leading to structures within the thorax (lung, heart) have been observed occasionally. The majority of these rare sequels of gastric disease are of pathological interest only. A few, however, may be benefited by surgical treatment. Their pathogenesis is much the same as in the case of the external fistulæ.

Gastro-colic Fistula.—Lieblein and Hilgenreiner collected 95 cases of this form of internal gastric fistula. The *cause* was recorded in 65 of the 84 cases studied by Chavannaz (1907): 47 were due to carcinoma (38 to gastric, 6 to colic cancer, the site in 3 cases not being recorded); 11 were due to ulcer (7 certainly, 4 only probably); 5 were the result of tuberculous disease of the stomach or colon; and 2 followed the formation of perigastric abscess. Its occurrence in connection with gastro-jejunal ulcer was mentioned at p. 391.

The *symptoms* are fecal vomiting and lienteric diarrhea. The ingested food, especially such articles as vermicelli, quickly appear in the stools and the similarity of the vomitus and the dejections is an important sign.

The *diagnosis* may be confirmed by roentgenological examination, or should this be unavailable, by distending the colon or the stomach

with air, and finding that the organ in communication becomes distended at the same time; or injection of colored fluids may be tried. Such tests are more apt to be successful when made through the bowel.

The *prognosis*, without operation, is gloomy. Chavannaz refers to two cases in which the fistula is said to have closed spontaneously, but neither patient was traced long afterward.

Palliative treatment consists in administering opiates or purgatives accordingly as there is diarrhea or constipation.

Surgical treatment has been undertaken in a number of patients. Six of the operations consisted, according to Chavannaz, in opening a perigastric abscess (2 patients), exploratory laparotomy (3 patients), or gastrotomy (1 patient). The operations of greater interest may be thus classified:

1. Colostomy. This operation is only palliative, and may be undertaken as a last resort to check fecal vomiting, by providing a false anus proximal to the gastro-colic fistula. The emaciation due to escape of food directly from the stomach into the colon would not be prevented. This operation was employed in one patient (Edmunds, 1884); no improvement resulted and death occurred 15 days later, the patient also having cancer of the rectum.

2. Jejunostomy. This operation is also merely palliative, but in cases of extreme emaciation would be of slightly more value than colostomy. It was employed by Labhardt and Eiselsberg (1901); the patient probably had cancer, but improved, and returned to his home one month later.

3. Separation of stomach from colon, with suture of orifices. This has been employed in a number of cases, of which the following four will serve as examples: (1) Fischer, 1888: resection of abdominal wall, of anterior wall of stomach, and of transverse colon; implantation of duodenum into remains of stomach, and fixation of both ends of colon in abdominal wound, for carcinoma. In spite of a secondary growth in the liver, noted at the time of operation, the patient recovered from the operation, lived five months, and died of carcinoma of the liver. (2) Henschel and Reichel, 1894: partial resection of gastric wall, and resection of portion of transverse colon, including entire lumen, for cancer. Operation lasted three hours and a half and ended fatally. (3) Czerny, 1902: separation of gastro-jejuno-colic fistula, occurring two months after posterior gastro-jejunostomy, with suture of the openings of the stomach and colon, and the performance of a new gastro-jejunostomy. Recovery. (4) Kauffmann, 1905, separation of the structures concerned in a gastro-jejuno-colic fistula (occurring four years after pos-

terior gastro-entero-anastomosis), with ileo-sigmoidostomy for stricture of transverse colon caused by suture of the two perforations in the colon. Time of operation, three hours. Six days later the abdomen was again opened for symptoms of perforation. These were found to be due to perforation of the sigmoid by the button used in making the anastomosis. The patient died on the table at the second operation.

4. Excision *en masse* of the diseased portions of stomach, colon and jejunum was done by Spassokukozki (1909), each viscus being repaired separately. His patient recovered, but symptoms of recurrence of the original pyloric ulcer soon developed.

5. Exclusion of the gastro-colic fistula. This may be either a complete exclusion, or a simple short-circuiting operation. (*a*) Complete Exclusion. Three such operations are referred to by Chavannaz. (1) Zweig and Hahn, in 1900, made an anastomosis between the transverse colon (above the fistula) and the sigmoid, and then occluded the colon on each side of the fistula by a purse-string suture. Time of operation, two hours and a half. Patient recovered and was in was in good health three years later. (2) Unruh and Garré, in 1899, divided the transverse colon above and below the fistula, closed all four ends of the colon, and then united the transverse colon above the fistula to the descending colon. The portion of the colon involved in the fistula was thus converted into a diverticulum of the stomach. The operation lasted two hours; the patient recovered, and was in good health 9 months later. (3) Kelling, in 1902, divided the transverse colon on both sides of the fistula, closed the ends of the gastric segment of colon by purse-string sutures, and did end-to-end anastomosis of the transverse colon. His patient recovered, but died 19 months later from cancer of the stomach.

(*b*) Short-circuiting was adopted in two cases mentioned by Chavannaz. Labhardt and Garré, in 1901, did a colo-colostomy of the transverse colon above and below the fistula. Their patient improved, and the fecal vomiting stopped. Chavannaz, in 1906, united the ascending colon to the sigmoid, for fecal vomiting due to cancerous fistula. The vomiting was not improved and the patient died in fifteen days.

It is interesting to note in this connection that Mauclaire (cited by Denéchau, 1908) purposely produced a gastro-colic fistula with the hope of relieving severe gastralgia which recurred one year after the performance of a posterior gastro-jejunostomy. On reopening the patient's abdomen, he found the gastro-jejunal anastomosis in good condition, and determined to do a gastro-colic anastomosis because, in a

previous similar case, no benefit had been derived from a second (anterior) gastro-jejunostomy. In the present patient the pylorus was thickened and indurated, but there was no open ulcer. The patient was much improved (!) by the formation of the gastro-colic fistula, but was not cured. Surely pylorectomy would have been better.

Jejuno-colic Fistula.—Gosset (1906) recorded a successful operation for this complication caused by the perforation of a peptic jejunal ulcer two years after posterior gastro-jejunostomy. He separated the jejunum and colon, sutured the perforations, and did ileo-sigmoidostomy because of the stricture thus produced in the colon.

VI. Duodenal Fistula.—This extremely fatal complication of upper abdominal lesions may follow either disease or trauma. Among the latter must be included operative injuries, such as are sometimes sustained in operations on the bile passages (Kraske, Kehr, Lilienthal, Mayo, Fink, Berg), or on the right kidney (Schede, Cackovic, Hitzrot, Thévenard). Lieblein and Hilgenreiner (1905) referred to sixteen cases of duodenal fistula, to which are to be added the cases subsequently reported by Berg (1907), Hitzrot (1910), and Thévenard (1913), all of which were traumatic in origin.

The *causes* are various. Perforation of an ulcer usually has produced a fistula above the bile papilla; and though operative fistulæ are said to be more often seen below this level, as are also fistulæ the result of rupture or gunshot wounds of the duodenum (see p. 307), yet in the authors' experience these post-operative fistulæ also have usually been above the entrance of the bile duct. The cutaneous orifice of the fistula is frequently in the right hypochondrium, but if the fistulous tract is retro-peritoneal, its outer opening may be in one of the intercostal spaces (Steaten, Gross), in the loin (Hinton), or even in the right inguinal region (Wagner); in Rintel's case the fistula was due to tuberculous peritonitis, and was at the umbilicus.

The *diagnosis* can be made usually with comparative ease, differentiation from gastric fistula, the only lesion with which it is readily confused, presenting few difficulties.

The *prognosis*, unless the fistula is a sequel of operation, is bad. Duodenal fistulæ following operation, whether from pressure of a drainage tube, from necrosis due to partial obliteration of the blood supply, or to an injury undiscovered during the operation, often heal spontaneously. But if the fistula is the result of duodenal ulceration, with subphrenic abscess or a long retro-peritoneal suppurating tract, emaciation is rapid, and if the patient does not die of starvation,

he is liable to do so from sepsis. Except for several post-operative cases, we know of no instance of recovery from duodenal fistula.

The only *treatment* which offers any hope of cure is operation. Traumatic lesions alone are suitable for suture (duodenorrhaphy); this was successfully adopted in one case by the senior author, in addition to posterior gastro-jejunostomy. When the fistula follows ulceration, and indeed also in many post-operative cases, much more may be hoped for from gastro-jejunostomy combined with unilateral exclusion of the duodenum by ligation of the pylorus. This operation was suggested by Cackovic and by Berg in 1903. Berg's first patient lived seventeen days; but in his second case he attempted to suture the perforation at the same time that he did gastro-jejunostomy; the sutures gave way, and then occlusion of the pylorus was performed too late to save the patient. The senior author has succeeded in bringing about healing of the wound in several cases by means of a pylorectomy including the duodenum down to a point below the fistula; this excision of course being supplemented by a posterior gastro-jejunostomy. By this technique all sutures are placed in healthy tissues, and there is little probability of the fistula again forming.

In patients with extreme emaciation some surgeons have done jejunostomy with the hope of doing a more radical operation should sufficient strength be gained. Cackovic (1903) tried this plan, but his patient died in two days. Direct feeding, either through the fistula, or through a duodenal tube introduced by mouth and allowed to pass the fistula, offers a better expectation of life; and if well tolerated, persistence in this latter method might even allow the fistula to close.

Internal Duodenal Fistula, *i.e.*, gastro-duodenal, duodeno-colic cholecysto-duodenal, etc., is so rare as to be of pathological interest only.

VII. Subphrenic Abscess.—This serious complication, though due to a variety of causes, is in such a large proportion of cases due to precedent gastric or duodenal disease, as to render it worthy of rather extended notice in this volume.

By the term subphrenic abscess is strictly understood only those abscesses formed between the dome of the diaphragm and some subjacent organ; but as usually employed the expression is made to embrace practically every abscess which at one portion or other of its circumference comes into contact with some portion of the diaphragm. Thus an abscess between the left lobe of the liver and the anterior gastric wall is included among subphrenic abscesses because of its close relation to the attachments of the diaphragm in the left epigastric and hypo-

chondriac regions; and one in the lesser peritoneal cavity will also be included because, by extending outward toward the spleen, or upward and backward along the Spigelian lobe of the liver, it will come into contact with the diaphragm in those situations.

The first description of subphrenic abscess, according to Freeman (1906), was that of Barlow, in 1845. In 1862 it was accurately described by Bouchard; and the first operation was recorded by Volkmann, in 1879. Maydl, in 1894, published a monograph on the subject based on a study of 179 cases. Finkelstein in 1899 collected 252 cases; Piquand (1909) collected 890 cases; and the subject was fully discussed by Barnard (1908), to whose excellent papers we are glad to acknowledge our indebtedness in preparing the present account.

A study of the pathogenesis of subphrenic abscess is considerably simplified by dividing the area beneath the diaphragm into certain definite anatomical regions (Fig. 15), as done by Barnard: "The under surface of the diaphragm is marked out into four peritoneal spaces and two cellular ones. The four peritoneal spaces are separated from one another by the cruciform arrangement of the ligaments of the liver—namely, the coronary, falciform, and the right and left lateral ligaments. The falciform ligament divides the subphrenic space into two parts, right and left. Each of these is again subdivided into a larger anterior and a smaller posterior part by the corresponding lateral ligament." The two cellular (extra-peritoneal) subphrenic areas are (1) that included between the layers of the coronary ligament; and (2) that extending from the cellular tissues around the upper pole of the left kidney up to beneath the left dome of the diaphragm. Barnard therefore classified the subphrenic fossæ thus:

A. Intra-peritoneal	I. Right	1. Anterior.	343 cases (Piquand)
		2. Posterior.	101 cases (Piquand)
	II. Left	3. Anterior.	300 cases (Piquand)
		4. Posterior	31 cases (Piquand)
B. Extra-peritoneal		5. Right.	132 cases (Piquand)
		6. Left.	19 cases (Piquand)

1. An abscess in the right anterior intra-peritoneal region is bounded above by the diaphragm, below by the right lobe of the liver, on the left by the falciform ligament; in front usually by adhesions between the hepatic margin and the anterior abdominal wall; while on the right such an abscess is frequently continuous with the right kidney pouch, from which direction the infection has most often travelled. Of 343 abscesses in this group, studied by Piquand, very few were due to perforation of gastric or duodenal ulcers; the great majority were

caused by appendicitis (77 cases), or by hepatic (61) and biliary lesions (50 cases).

2. The right posterior intra-peritoneal region is continuous, between the posterior surface of the right lobe of the liver and the lower ribs, with the right kidney pouch and subhepatic fossa. In its strictly subphrenic region such an abscess would be bounded above by the diaphragm, below by the upper surface of the posterior portion of the right lobe of the liver, in front by the right lateral ligament, and on the left by the reflection of parietal peritoneum covering the right surface of the vena cava and becoming continuous between diaphragm and liver with the coronary and right lateral ligaments of the liver. Abscesses in this situation also are commonly due to infection through the right renal pouch, and thus such an abscess may sometimes extend beneath the right lobe of the liver, between it and the transverse mesocolon, across the foramen of Winslow (which easily is occluded by plastic peritonitis) to the anterior surface of the gastro-hepatic omentum, where it will be bounded above by the left lobe of the liver, below by the stomach, in front by the abdominal wall and the diaphragm. Of course, it is also possible, though less usual, for infection to travel in the other direction, starting on the anterior surface of the stomach, passing to the subhepatic region, up the posterior abdominal wall, to become again finally subphrenic. This area is thus seen to be very large and irregular in outline: it includes not only the region above the right lobe of the liver, back of the right lateral ligament, but also the subhepatic space, which has its base in the lateral abdominal wall, and its apex between the left lobe of the liver and the anterior gastric wall. Among Piquand's 890 cases of subphrenic abscess, this region was involved only in 101 cases; and in only 47 of these was the abscess limited strictly to this space.

3. Abscess in the left anterior intra-peritoneal region is bounded above by the diaphragm, below by the left lobe of the liver, on the right by the falciform ligament, on the left by the spleen, posteriorly by the left lateral ligament, and anteriorly by adhesions between the anterior surface of the stomach, the transverse colon, the great omentum, and the abdominal wall. Below the anterior margin of the left lobe of the liver this pouch is continuous on the right with the subhepatic pouch, and on the left with the lumbar pouch, from which infection may reach it by travelling between the spleen and the splenic flexure of the colon. This left anterior intra-peritoneal sub-diaphragmatic region is that which is most often invaded by gastric ulcers which perforate subacutely. Among Piquand's cases, there were 300

instances of suppuration in this space, and 138 of these abscesses were due to gastric perforations.

4. An abscess in the left posterior intra-peritoneal region arises in the lesser peritoneal cavity. The Spigelian lobe of the liver, which lies in the roof of this cavity (see p. 19), is in contact with the peritoneum covering the left crus of the diaphragm. This portion of the lesser peritoneal cavity is usually the last to be invaded, so that frequently a so-called subphrenic abscess in this region will have no direct relation to the diaphragm. The lesser peritoneal cavity of course communicates through the foramen of Winslow with the subhepatic pouch; but, as is well known, inflammatory processes in this region tend to become encysted by the early obliteration of this foramen by adhesions. It is very rare for an abscess to spearate the layers of the great omentum and form a secondary omental abscess. The most usual cause of suppuration in the lesser peritoneal sac is perforation of a gastric ulcer. Among Piquand's 890 cases there were 31 abscesses in this situation: 13 were due to pancreatic lesions. Michel and Gross in 1904 collected 44 instances of suppuration in the lesser peritoneal cavity. They classified the causes thus: 1. Encysted hemorrhage which has become infected, due originally to pancreatitis hemorrhagica or to trauma. 2. Directly from pancreatitis. 3. Perforation of the stomach. 4. Diseases of the spleen. 5. General peritonitis. The junior author reported (1902) a case of gastric ulcer in which perforation occurred nearly simultaneously on both anterior and posterior walls, producing two subphrenic abscesses, one beneath the left lobe of the liver, anterior to the gastro-hepatic omentum, and the other in the lesser peritoneal cavity.

5. An abscess in the right extra-peritoneal subphrenic region lies between the layers of the coronary, the two lateral, and the falciform ligaments of the liver. This form is due mostly to abscess of the liver or other forms of hepatic disease; other recognized causes are affections of the right kidney, retro-peritoneal appendicular suppurations, retro-peritoneal duodenal perforations, and occasionally perforation of the diaphragm as the result of thoracic disease. Such abscesses may point in the epigastric region, or rarely at the umbilicus.

6. The left extra-peritoneal subphrenic region is usually infected through the structures around the spinal column or the left kidney. Two of Barnard's cases were due to acute periosteitis of the transverse vertebral processes, a third was caused by an empyema, and "the fourth was probably due to a posterior perforating gastric ulcer," as in the similar case described by Robson.

Cause.—As may be seen from the preceding paragraphs, the causes of subphrenic abscess are many and varied. The statistics of Lance (1909), comprising almost a thousand cases, indicate that about 20 per cent. are caused by appendicitis; 30 per cent. by lesions of the stomach and duodenum; 13 per cent. by lesions of the liver and gall-bladder; and 37 per cent. by miscellaneous affections (pancreas, spleen, large intestine, pleura, etc.).

Pathogenesis.—We may then briefly study the origin and method of infection in these various classes.

Gastric ulcers most often perforate anteriorly. If diffuse peritonitis is not at once produced, the perforation will be subacute, protective adhesions having quickly formed. Under these circumstances the resulting abscess will occupy the apex of the subhepatic fossa, between the left lobe of the liver above, the gastro-hepatic omentum posteriorly, and the stomach below. Unless quickly relieved, such an abscess is prone to leak, producing secondary diffuse peritonitis, usually of the progressive fibrino-purulent form described by Mikulicz. If neither leakage nor rupture occur, the pus usually will work its way around the anterior margin of the left lobe of the liver, invade the left anterior subphrenic space, and form a true subphrenic abscess. Although this anterior perigastric abscess is almost invariably the result of subacute perforation of a gastric ulcer, it may occur on the subsidence of an unoperated diffuse peritonitis due to acute perforation. Such a case was observed by the junior author under Dr. T. R. Neilson's care at the Episcopal Hospital (1908). Perforation of a posterior gastric ulcer causes suppuration in the lesser peritoneal cavity; very rarely has perforation on the extraperitoneal surface of the stomach led to subphrenic abscess (J. A. C. Macewen, 1920). Eliot (1912) has reported a case of bilateral subphrenic abscess due to duodenal ulcer. Sequels of these epigastric abscesses other than fatal peritonitis are rare; but among the results which are occasionally seen may be mentioned the various forms of gastric fistulæ, already described (p. 397); and perforation of the pleura, the lung, the pericardium, etc.

Duodenal ulcers on perforation infect either the right anterior subphrenic region, if intra-peritoneal, or the right retro-peritoneal cellular tissue, if perforation occurs on the posterior or internal surface of the bowel. Intra-peritoneal infection has a marked tendency to gravitate to the right lumbar region, and by invading even the iliac fossa may simulate appendicitis.

Appendicitis itself may give rise to subphrenic abscess in various ways. Elsberg (1901) collected 73 such cases, and to these Eisendrath

(1908) added 33 others, including 5 of his own. It occurred in 20 out of one series of 2400 cases of appendicitis under the care of the senior author, 4 patients recovering. Intra-peritoneal subphrenic abscess is much more often a complication of appendicitis than is extraperitoneal. Among the 106 cases analyzed by Eisendrath, the abscess was intra-peritoneal in two-thirds. In the usual variety the right renal pouch is first affected, then the right posterior intra-peritoneal subphrenic space, including the subhepatic space, and finally the right anterior subphrenic space may be invaded around the right free extremity of the lateral ligament of the liver. It is a mistake to assume, as was done by Ross (1911), that every appendicular abscess in the right kidney pouch or subhepatic space is to be classed as a subphrenic abscess. If the appendix lies to the inner side of the colon, in front of the mesentery of the ileum (a very unusual position), the apex of the subhepatic space may be infected directly, without involvement of the right renal pouch. Extra-peritoneal subphrenic abscess as the result of appendicitis may occur by continuity of tissue, or secondarily through invasion of the liver after suppurative pylephlebitis. The same course of events may of course occur as the result of gastric disease. Eisendrath found recorded only six left-sided cases of subphrenic abscess due to appendicitis.

Hepatic abscess frequently becomes subphrenic by the process of pointing of an abscess through the convex surface of the liver.

Diagnosis.—According to Barnard, in aiming to arrive at a diagnosis in cases of suspected subphrenic abscess, special attention should be paid to the following points:

1. *The Previous History of the Patient.*—The usual *causes* of the condition, e.g., gastric or duodenal ulcer, appendicitis, hepatic abscess, dysentery, etc., must be studied.

2. *The Character of the Onset* is important. If the symptoms were acute, the abscess probably is intra-peritoneal; but if insidious in their origin it is more likely to be situated extra-peritoneally, or in the lesser peritoneal cavity.

3. *The Constitutional Signs of Pus* should be searched for: elevation of temperature, persistent, even if slight; chills; emaciation; thirst; leukocytosis; etc. Other possible regions of suppuration should be excluded.

4. *Abdominal Signs and Symptoms.*—These include bulging, immobility during respiration; tenderness, rigidity; dulness, or tympany due to the perforation of an air-containing viscus. A swelling due to subphrenic abscess is immobile because fixed by adhesions.

5. *Thoracic Signs and Symptoms.*—These were present in 56 out of 76 cases of subphrenic abscess studied by Barnard. The most important are: dulness, associated with upward displacement of the lung; diminution or absence of breath sounds, vocal resonance, and vocal fremitus. Occasionally dulness on percussion may be associated with tubular breathing and increased vocal resonance. Amphoric resonance, the coin sound, etc., may be present in abscesses containing air. The apex beat of the heat may be displaced upward, but seldom laterally. Hoover (1913) lays stress on relative increase or decrease of the excursion of the costal border on the affected side, in differentiating between subphrenic abscess and empyema: if the abscess is subphrenic the diaphragm is so much arched that it acts at a disadvantage and the other respiratory muscles (chiefly the intercostals) cause the excursion of the costal margin to be increased; the reverse is true (that is the excursion of the costal margin on the diseased side is decreased as compared with the healthy side) if empyema is present and the diaphragm is depressed to a plane surface. However, in cases of massive empyema the diaphragm may become concave superiorly; in such a case this sign no longer holds good.

6. *Localizing Signs* should be looked for as an aid to operation. They embrace bulging; tenderness; increase in circumference of the lower thorax on the side affected; edema; enlargement of the veins, etc. *Fluoroscopic Examination* shows fixity or lessened mobility of the diaphragm on the affected side.

7. *Aspiration* is dangerous unless followed by immediate operation when pus is found; on the other hand, failure to find pus by no means excludes the presence of an abscess, but may cause the postponement of an operation until it can no longer be of benefit. Hence the needle should not be used until the patient is on the operating table ready for any operation that may seem proper.

Prognosis.—The prognosis of subphrenic abscess is bad, no matter what the treatment; but it is very much worse if no operation is done, or if operative treatment is too long delayed. Among Barnard's cases there were 64 patients treated by 73 operations; of this number 40 patients recovered, and 24 died, a general mortality of 37.5 per cent. Death resulted in every patient not operated upon. Among the 44 cases of suppuration in the lesser peritoneal cavity collected by Michel and Gross, there were 19 patients treated by operation; all of those not operated upon died, while of the others only 9 died, a death rate of 47.36 per cent. Of the patients with subphrenic abscess following appendicitis, studied by Eisendrath, 84 were treated by operation with 23 deaths, a mortality of 27.38 per cent.; while the death rate

among patients not operated upon was over 82 per cent. As already noted, there were 16 deaths among 20 patients under the senior author's care, a mortality of 80 per cent.

Barnard concluded that, speaking in a general manner, posterior methods of drainage give more favorable results than do the anterior. In his series of cases, 26 posterior operations were performed, with 7 seven deaths (27 per cent.); 43 anterior operations, with 17 deaths (39.5 per cent.); and 4 lateral, with 3 deaths (75 per cent.). He had himself performed 24 operations on 21 patients for subphrenic abscess; 15 of these operations were posterior, with 2 deaths (13.3 per cent.); and 9 were anterior, with 2 deaths (22.2 per cent.).

Treatment.—The operations for subphrenic abscess may be classed in accordance with Barnard's tables, as anterior, posterior, and lateral. The former include abdominal incisions, whether in the epigastrium or in one of the hypochondriac regions. The posterior operations include the subpleural and transpleural approaches through the diaphragm; they resemble the usual operations for hepatic abscess, which will be described in connection with that lesion in a subsequent chapter (p. 791). Lateral transpleural or subpleural operations should not be attempted unless the abscess is very manifestly pointing in the axillary line.

When the existence of subphrenic abscess is suspected, it is sometimes better, as pointed out by Barnard, to delay operation for three or four days in order to allow the abscess to become more accessible. This applies particularly to infections beneath the right dome of the diaphragm, secondary to disease of the liver. When the abscess is believed to be in other situations we believe delay to be dangerous, and think an exploratory laparotomy should be undertaken as soon as the presence of pus is reasonably certain, even though its exact location cannot be pre-determined. By opening the peritoneal cavity and cautiously disposing gauze packs before attempting any exploration whatever, or before rupturing any adhesions, it usually will be possible to discover the situation of the abscess, and then to approach it by the thoracic or lumbar route should such a course be advisable. Especially dangerous is delay in those subphrenic or perigastric abscesses which arise as the localized remains of a diffuse peritonitis (residual abscesses). In such cases the limiting adhesions are never so firm as in subacute or chronic perforations, and unless the abscess be evacuated so soon as its presence is detected, it will be sure to break again and produce spreading fibrino-purulent peritonitis, from which very few patients will recover.

The use of the exploring needle may be preferred to laparotomy

when the patient, prepared for operation, is on the operating table, and the surgeon has good reason to believe that the abscess is beneath the costal margin. Attention to the known pathogenesis of subphrenic abscess will frequently enable this point to be determined with reasonable certainty.

In all cases in which the abscess has to be approached by the anterior (abdominal) route it will be much safer for the surgeon to open it transperitoneally after thoroughly protecting all surrounding structures by gauze packs, than for him to attempt to cut directly into the abscess cavity. In the latter method of operating the surgeon can never be entirely sure that his incision itself has not trespassed beyond the limiting adhesions, nor that his manipulations have not produced leakage into the general peritoneal cavity at some other point of the abscess's periphery.

For *anterior perigastric abscess*, median or left hypochondriac laparotomy is to be preferred. Drainage may be provided for by a counter opening in the left flank, if the abscess extends far toward the spleen.

For *posterior perigastric abscess* (suppuration in the lesser cavity of the peritoneum), laparotomy should be done, and the abscess opened where most prominent—whether it points through the gastro-hepatic or through the gastro-colic omentum, or through the transverse mesocolon. Though recovery has followed anterior drainage alone, it is much safer to make a counter opening in the left loin, below the last rib; under such circumstances it is occasionally proper to close the anterior incision without drainage. If the location of the abscess in the lesser peritoneal cavity has been determined before operation, an attempt should be made to open it through the left ilio-costal space. This route has been employed several times successfully by the senior author, either as the primary operation, or after locating the abscess by laparotomy.

For a subphrenic abscess which involves the *subhepatic space* and right renal pouch, laparotomy combined with counter-drainage in the right loin should be employed.

When the abscess does not extend beyond the confines of the costal margin on the right it is sufficient to drain it by the thoracic incision.

The thoracic operation should never be employed if there be diffuse peritonitis. In the presence of this additional complication there is a choice of two methods of procedure: 1. If it appear that the patient will survive the immediate shock of an operation, laparotomy should be done, the cause of the peritonitis abated, and drainage of the pelvis

provided for, as well as of the side of the subphrenic space involved. 2. If the peritonitis has advanced so far that no operation can be undertaken without great probability of hastening the patient's death, the starvation treatment of Ochsner should be adopted; but any localized collection of pus must be opened as soon as it is discovered—delay of even twelve hours may place the patient beyond the reach of surgery. Whichever plan of treatment be adopted, the head of the patient's bed should be raised 30 degrees from the floor (Fowler's position), to aid the gravitation of septic fluid to the pelvis. Water should be constantly administered by the bowel.

PART II

CHAPTER XVI

SURGICAL DISEASES OF THE BILIARY TRACT

General Etiological, Pathological and Diagnostic Considerations

The prime factor in all non-neoplastic surgical diseases of the biliary tract is some form of micro-organismal invasion. The consequences of this may be slight or grave, transient or chronic, accompanied by very slight or very decided structural changes, the formation of concretions, etc.; but invariably, we believe, the underlying, exciting cause has been infection of the tract by bacterial life. The only exception to this statement may be found in those rare instances where surgical diseases result from the catarrhal condition of the bile-passages consequent upon the action of some toxic substance, such as is seen in acute phosphorus poisoning.

Our present knowledge of the bacteriology of the biliary tract is the result of the work of numerous investigators.[1] It is known that when bacteria gain entrance to the biliary tract they may give rise to infections that result in (1) a mild catarrhal condition of the tract; (2) acute suppurative cholangeitis with or without empyema of the gall-bladder; (3) mild, or severe, gangrenous cholecystitis; (4) gall-stone formation, etc. It is known that the bile itself is a medium for the growth of many forms of bacteria, and that it is not, as was formerly believed, a bactericidal agent.

Bacteriology of the Biliary Passages.—It generally has been claimed that normal bile in normal bile-passages is sterile (Hoppe-Seyler, 1903). On the other hand Lippman (1904) demonstrated bacterial life in apparently normal common ducts and in the normal ampulla of Vater; while the researches of Bond (see page 417), prove conclusively that it is possible to find bacteria in normal mucous ducts.

In our own experience cultures from the biliary tract taken at operation proved sterile in over 55 per cent. of the cases examined. This is to be explained by the fact that a majority of operations are done not

[1] Free use has been made, in what follows, of the Mütter Lecture of the College of Physicians of Philadelphia (1905), by Dr. A. O. J. Kelly, late Pathologist to the German (now Lankenau) Hospital.

during the acute stage of bacterial invasion, but for the remote results of the infection (gall-stones): in the interval the biliary tract has cleared itself of the bacteria, though the latter may in some cases still be recovered by culture from the interior of the calculi even when the bile itself is sterile.

The bacterium most frequently found in the biliary tract is the Bacillus coli communis, which was found in 68 per cent. of our non-sterile cases; it was followed in the order of frequency by the Bacillus typhosus (over 15 per cent.).

Among other bacteria that have been found in bile may be mentioned Staphylococcus, Streptococcus, B. subtilis, and numerous others.

Avenues of Infection.—Bacteria gain entrance to the biliary tract, in most instances, through the *portal circulation*, or through the *general circulation*. Invasion also takes place through the duodenal opening of the *common bile-duct*, through the *lymphatic circulation*, and possibly through the *walls of the biliary tract*. Of these various avenues of entrance the portal circulation is the most frequently traversed, although many investigators have claimed that the ductal opening in the duodenum is the chief gateway of entrance. It must be remembered that the duodenum, in a normal condition and especially when free from food, is almost free of germ life. Kelly pointed out two other important factors that must be considered in connection with the entrance of germs through the ductal opening: one is the sphincteric action of the ampulla of Vater, which Archibald (1912) found exerted a force equivalent to about 600 mm. of water pressure within the duct;[1] the other is the peculiar flushing of the duct and its opening by the intermittent gushing of bile into the duodenum. Were there a constant, free opening of the ampulla, with a slow steady flow of bile through the opening, this entrance would be a more common avenue of infection than it is. During the presence of food in this portion of the intestine, which is almost the only time germs are present in the normal duodenum, there is an intermittent expulsion of bile (see p. 38) which naturally clears the opening and the first part of the duodenum of all bacteria that may become lodged there.

The comparative freedom of the pancreas from infectious diseases has been advanced as a strong argument against invasion taking place through the ampulla of Vater. But even if the bacteria make no selection between the pancreatic and the common bile-ducts when they reach the crossroads where these two channels join, those entering the pancreatic duct are confronted by much greater obstacles to over-

[1] Oddi (1888) found it equal to 675 mm. of water pressure.

come than are their fellows who enter the bile channel. Many claim that the pancreatic juice is antagonistic to germ life, while the bile, especially when stagnant, stimulates instead of hinders the growth of many forms of bacteria. There is no such pool of stagnant pancreatic juice as there is of bile in the gall-bladder, and hence no suitable lodging-place where bacteria may thrive and multiply.

Bacteria have, however, been found in the normal diverticulum and in the lower end of the normal common duct. It is of course possible that such bacteria may have descended with the bile from the liver, which is constantly inundated by bacteria through the portal circulation. But the experimental work of Bond (1905), showed that it is possible for micro-organisms to travel against the natural currents in mucous ducts: He was able to recover through a fistula into the gall-bladder granules of indigo blue which had been administered by mouth. His experiments seem to prove conclusively that it is perfectly possible for bacteria to enter the common duct from the duodenum and traverse its length. This may be accomplished not only by the motile bacteria, such as the colon, typhoid, and paratyphoid bacilli, but also by the non-motile staphylococci and streptococci.

The usual pathway of entrance in cases of infection of the biliary tract is through the *portal circulation.* Adami has shown that under normal conditions bacteria may be found in the deeper layers of the intestine, in the portal circulation, and in the liver. He advances the theory that the leukocytes carry the bacteria to the lymphatic radicles and also to the radicles of the portal vein.[1] They may also be taken up by the thoracic duct and emptied into the general circulation. The natural resistive and bactericidal powers of the liver cells are sufficient to overcome and destroy most of the germs arriving through the portal circulation. If there should be a weakened condition of the hepatic cells, however, either through disease or overwork, many of the bacteria will escape destruction and be excreted with the bile. The researches of Lartigau (1906) definitely settled the fact that infection of the biliary tract may be a descending one from the liver. Having tied the common duct in animals, Lartigau fed them bacteria, especially the Bacillus pyocyaneus, and was able to recover this germ from the gall-bladder in almost 50 per cent. of the animals so treated. In many instances the Bacillus pyocyaneus was recovered in pure cultures; in other instances the colon bacillus was associated with it.

Infection of the biliary tract through the *systemic circulation*

[1] It is in this way that *appendicitis, typhoid fever* and other infections in the abdomen may act as a predisposing factor for infections of the biliary tract.

is possible and, in many instances, probable. The advancement in the study of the blood which has made it possible to determine the presence of bacteria in the general circulation in certain infectious diseases, such as typhoid fever, has made the systemic circulation seem much more important as a pathway of entrance for bacteria to the biliary tract. The work of Dörr (1905) proved that bacteria injected into the circulation of a rabbit, can be recovered from the gall-bladder in a few hours. As pointed out by Kelly the infections of the biliary tract so often found as complications of systemic infections also show the possibility and probability of infection through the blood. It cannot be stated definitely, however, whether the infection, in these cases, takes place through the hepatic and cystic arteries, through the portal vein, or through the common duct from the intestinal canal. The presence of typhoid bacilli, unassociated with other bacteria, in the gall-bladder in cases of enteric fever naturally strengthens the belief that in some instances at least the systemic circulation conveys the germ to the biliary tract. This belief is confirmed by the experiments and findings of Joseph Koch (see page 428), as well as those of Wrzosek and of Else (1910) and by the more recent investigations of Rosenow (1914) who claims that bacteria exert a selective action in choosing their place of lodgment, certain strains being specific for the gall-bladder, others for the pylorus, the appendix or other structure.

In exceptional cases infection of the biliary tract may take place through the *lymphatic system*, as was probably the case in an instance reported by Müller (1905). Bishop (1907), also, claimed that infection may take place through this channel, although its occurrence is very rare.

Infection through the walls of the biliary tract, and especially through the walls of the gall-bladder, may occur by *contiguity*. It is questionable, however, whether the infection travels from without inward in those cases where the gall-bladder is surrounded by a mass of adhesions; it is much more likely that they are relics of a former inflammatory condition which probably had its origin within the bile passages. Under such circumstances any new inflammatory processes are more likely to be a relighting of the old infection rather than a new one.

Pathogenesis.—After the micro-organisms have entered the biliary tract the consequences of their activity will vary with the virulence of the invading bacteria, the presence or absence of calculi, the resistance offered by the structures affected, and the anatomical peculiarities

of the portion of the tract involved. These changes may vary from a very mild, quickly subsiding acute infection, to the most violent, fulminating, gangrenous process; from a mild, almost symptomless catarrh to a chronic inflammation of the biliary tract which may last for years, with acute exacerbations causing greater structural changes with each attack. Or the tract, and especially the gall-bladder, may be utilized as a store-house for masses of bacteria, as frequently demonstrated in the cases of "chronic typhoid carriers" (see page 432).

In many instances the only changes that can be noted are a slight swelling of the mucous membrane with possibly some slight exfoliation of the epithelium. Such patients usually rid themselves of the infection through their natural resistance and by an increase in the flow of bile which washes the germs from the tract, thus allowing the affected area to heal with only trifling change of structure. With infection of greater virulence, or where lessened resistance exists, an acute inflammation of the ducts or gall-bladder or both, with or without pus formation, will be found.

The presence of inflammation in the ducts or gall-bladder will cause the mucous membrane lining the cystic duct to become swollen with consequent narrowing of its lumen and damming of bile behind the obstruction. The partial or complete stasis of bile thus produced provides an opportunity for germs to exert their full power, the result being determined by the virulence of the micro-organisms present, and the ability of the gall-bladder and ducts to resist and overcome the infection.

Gall-stone Formation

Historical.—Many and various theories have been advanced at different times to account for the formation of gall-stones. A more exact knowledge of the anatomy of the biliary tract, with a better understanding of the physiological action of the liver and the physiology and chemistry of the bile, enhanced as these have been by the pathological findings at the operating table, have explained the most important parts of the subject. The work of Naunyn settled the question definitely in the opinion of his followers. The researches of Kramer (1907), of Bacmeister (1908), and of Bishop (1907), however, threw doubt on some of Naunyn's findings. A few questions concerning gall-stone formation remain unanswered, although they are not of great importance.

Originally, gall-stones were supposed to be formed from coagulated bile, the coagulation being caused by a rise in the temperature of

the liver.[1] Paracelsus believed that gastro-intestinal disturbances caused an acid condition of the blood, the acids which were formed acting upon the bile with consequent precipitation and formation of concretions. Later as a better knowledge of the normal constituents of bile was obtained, it was thought that a super-abundance of any of its various elements might exist, with consequent stone formation. Meckel called attention to a chronic catarrhal condition of the mucous membrane of the bile-tract and especially of the gall-bladder, and advanced the theory that a "stone-forming catarrh" might be the basis of the lithiasis. Flint suggested that excessive brain work caused a condition of cholesteremia; this gave rise to the theory that gall-stones were formed as soon as an excessive amount of cholesterin was present in the blood. Hein thought that plugs of mucus, the product of a diseased condition of the mucous membrane, acted as nuclei around which the stones were formed. He also claimed that changes taking place in the bile within the bile-tract might cause the formation of stones—small, minute, bile-pigment stones first being formed in the ducts; and later, after they had reached the gall-bladder, acting as nuclei for larger stones. The supposition that the epithelial cells of the gall-bladder were important factors in gall-stone formation was suggested by Seifert. Naunyn (1892) advanced the idea that most stones were formed within the gall-bladder as a result of a catarrhal condition of the mucous membrane consequent upon bacterial invasion, desquamation of epithelial cells, and deposition from these cells of larger quantities of cholesterin than the bile could hold in solution. He also claimed that the calcium bile-salts acted as a cement substance, through the action of which accretions were constantly added to the original nucleus or stone. Naunyn also thought that the bile-pigment stones were formed in the ducts through oxidation of bile, this being brought about by the action of bacteria. Naunyn's belief is generally accepted to-day, although later investigators differ with him as to the origin of the excess of cholesterin.

Pathogenesis.—The actual formation of gall-stones is caused by an increase of the *cholesterin* constituent of the bile, a deposition of *calcium bile-salts*, especially bilirubin-calcium, in the presence of a *stasis or sluggishness in flow of bile*. The increase in cholesterin and the deposition of the calcium salts are due to the action of bacteria. This was shown by the experiments of Gérard (1905) and of Bacmeister (1908). Neither of these observers was able to produce concretions in

[1] Historical references are given in the monograph of Exner and Heyrovsky (Archiv. f. klin. chir., 1908, lxxxvi, 609).

sterile bile contained in a test-tube, but when colon bacilli were present deposition of salts readily ocurred. The interference with the natural flow of bile also may be due to the action of bacteria in creating a catarrhal inflammation of the mucous membrane of the gall-bladder and ducts; or it may be consequent upon any of those predisposing causes of cholelithiasis (see page 476) which have a tendency to cause stagnation of bile. It is necessary for the inflammatory changes caused by the bacteria to be of a very mild character; the bacteria themselves must be almost non-virulent. A virulent inflammation of the gall-bladder or bile-tracts will cause rapid changes in the structures involved, acute cholecystitis with its train of complications and sequels (see page 449), a cholangeitis of purulent character, etc., rather than the mild, almost symptomless inflammation that is necessary for gall-stone formation.

The source of the increased amount of *cholesterin* is a question that has not been definitely settled. Naunyn (1905) attributed it to the cells of the mucous membrane lining the biliary tract which are exfoliated in excessive numbers as a consequence of a very mild, catarrhal inflammation. These cells contain droplets of myelin, of fluid cholesterin. Kramer (1907) on the other hand, derived results from test-tube experiments which make it very probable that the cholesterin may be deposited from the bile itself. He was able to obtain a precipitate from a culture medium (consisting of equal parts of bouillon and bile) which had been inoculated either with the colon bacillus or the typhoid bacillus. The precipitate increased with the age of the culture. Microscopically the precipitate consisted of biliary coloring matter, cholesterin, magnesium phosphate, calcium phosphate, calcium carbonate, and bacilli. Kramer concluded from these experiments that "gall-stone formation is due to a chemical decomposition of bile, the direct result of the growth of micro-organisms therein." Bacmeister also differs from Naunyn regarding the source of cholesterin, and from Kramer as to the direct cause of its deposition from bile. He claims that there is a more or less constant autolysis of the bile, with the deposition of cholesterin from it; that this autolysis is much more marked when the bile is contaminated by epithelial cells or bacteria. Experimentally, he was able to demonstrate the separation of cholesterin crystals from sterile bile, and even from filtered bile, although separation in the latter instance was much slower than in unfiltered bile. In the presence of the Bacillus pyocyaneus, the Bacillus proteus, the Bacillus typhosus, or the Bacillus coli communis he was able to demonstrate a very marked precipitation of cholesterin. Bishop holds that an inflammatory condition of the mucous membrane of the

biliary tract is the initial step in gall-stone formation, the change in the membrane being the direct result of bacterial invasion, the bacteria penetrating the walls of the tract with the resultant mild inflammation. The result of this inflammation is an outpouring of cholesterin in larger quantities than can be dissolved by the bile. This view is in keeping with that of Naunyn and was well summarized by Kelly (1908) who said "the increased cholesterin, then, is derived not from the bile," "it results from catarrhal disintegration of the mucous cells lining the wall of the gall-bladder" (the seat of the formation of most gall-stones).

All observers are agreed that the mild inflammation of the mucous membrane causes an increased secretion of mucus, an albuminous substance which causes precipitation of the *calcium bile-salts*, especially the bilirubin-calcium. There is also an increase in the exfoliation of epithelial cells. Naunyn claims that the mucus, cholesterin and epithelial cells act as a nucleus for the beginning stone, being held together by the bilirubin-calcium which acts as a cement substance. Bacmeister, on the contrary, claims that the first stone formed is composed, almost invariably, of pure cholesterin; that the presence of this stone creates conditions very favorable for bacterial infection with inflammatory changes in the walls of the gall-bladder, and that these changes cause marked increase in the secretion of mucus. This mucus is very rich in calcium salts which furnish material for the formation of new stones, as well as for the deposition of additional layers on the surface of the stone already formed. Basing their views largely on the experimental work of Bacmeister, Aschoff and Bacmeister, in their monograph on Cholelithiasis (1909) claimed that with very few exceptions the first calculus—a cholesterin stone with radially disposed crystals—is formed without any bacterial infection of the biliary tract, merely by chemical changes in the stagnant bile of a gall-bladder more or less obstructed by mechanical means, such as kinks of the cystic duct, a viscid state of the bile, etc.

The *sluggishness in the flow of bile*, or even actual *stasis* is favored by two principal factors: (1) changes in the bile itself; (2) changes in the bile-ducts. The composition of the bile is altered by the chemical changes already alluded to, induced by bacteria; it becomes thicker, more viscid, less fluid. The bile-ducts become narrowed and eventually obstructed by inflammatory edema, by kinks due to the drag of an inflamed gall-bladder, by impaction of calculi, or by pressure of surrounding adhesions, tumors, etc. Waugh (1920) has made a strong plea for the recognition of a congenitally mobile ascending colon as a factor in

the production of the stagnant gall-bladder. In ten patients in whom these lesions were associated he noted the presence of a well developed fold of peritoneum running from the fundus of the gall-bladder to the hepatic flexure of the colon (the so-called cholecysto-colic ligament), which he regards as the medium of traction.

Moreover, when calculi have once formed, the impaction of a calculus, and the damming up behind it even temporarily, of altered bile, must be recognized as favoring the formation of other calculi.

Finally, a word should be said about calculous formation around *foreign bodies* as a nucleus (page 479). Such cases have been observed by many surgeons, including Homans (1897) and Kehr (1901), each of whom reported instances of stones forming around silk ligatures employed at operation. Hence the rule, in biliary surgery, to employ only sutures of absorbable material. It has been shown experimentally that sterile foreign bodies introduced into the healthy gall-bladders of dogs do not lead to the formation of gall-stones (Meyer); but that in the presence of attenuated infection concretions may readily be produced (Mignot).

The *rapidity of gall-stone formation* is much greater than commonly supposed. Naunyn, in 1905, stated his belief that they might be formed within a few hours. Aschoff and Bacmeister claim, however, that the radial cholesterin stone is of extremely slow formation, many years being required to produce one of nut-size.

Physical Characters of Gall-stones.—The *color* depends upon the composition of the stone, varying from the almost pure white or yellowish stone, which is composed of cholesterin, to the black stone which contains an abundance of bile-coloring matter. The color varies in all stones, even the practically pure cholesterin stone showing dark patches of coloring matter between the crystals, and no stone being uniform throughout. The surface is generally darker than the inside of the stone; while the nucleus is, as a rule, lighter in color than the surrounding parts.

The *hardness* of the stone varies in proportion to the amount of calcium salts present. The cholesterin stones, while more or less firm and hard to the touch, are brittle and can readily be broken between the thumb and fingers. Stones recently formed generally are soft.

The *shape* of the concretions generally varies with the number present. Single stones are more or less rounded in shape, and of course, never faceted. When a number of stones are present, they are usually faceted, being pressed into this shape by the contractions

of the gall-bladder while they are still soft (Fig. 132). As a rule, a large mass of stones, unless greatly disturbed, will take the form of the gall-bladder, the portions of the stones on the outside of the mass and in contact with the walls of the gall-bladder being usually rough and more or less rounded, while the portion of each stone in contact with other stones will be pressed flat. The stones in the center of the mass are almost universally faceted. Naunyn asserts that this faceting is due entirely to pressure, and not to a grinding motion among the stones; but it is difficult to see how some of the facets become so highly polished unless it is by attrition.

The "mulberry" stones generally are composed of a great number of small concretions which have coalesced, finally being covered with a new layer which gives the stones their appearance of uniformity.

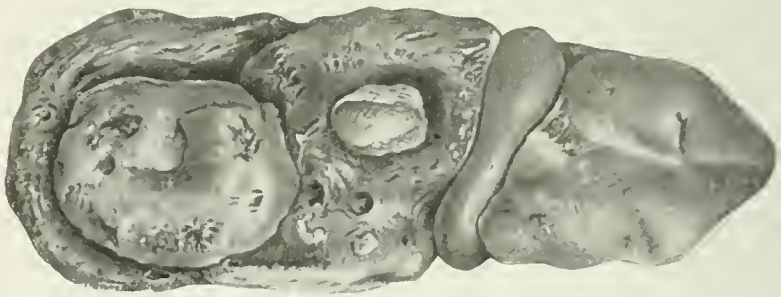


FIG. 132.—Mass of Gall-stones Conglomerated and Pressed into the Shape of the Gall-bladder. From a Patient in the Lankenau (Formerly German) Hospital.

In *weight* and *size* the largest stone known is that reported by Richter in 1793: this calculus, removed from the common duct at autopsy, weighed three ounces and five drachms (116 grams). W. Bartlett, of St. Louis, has successfully removed from the common duct a stone measuring 4 by $1\frac{1}{2}$ inches, and weighing two ounces and a half (80 grams). For other noteworthy instances of unusually large stones, the reader may consult Packard's monograph (1903).

The size of the stones usually varies inversely with their number, which may range from a single stone to many thousands. There seems to have been nothing discovered which will definitely account for their variation in number. The number formed may depend upon the amount of calcium salts present. These act as a cement substance; and when they are present in abundance, there may be a coalescence of a greater number of the particles of mucus, cholesterin and epithelial cells which generally form the nuclei, and a correspondingly smaller number of fully developed calculi; while with a deficiency

in the calcium salts, the particles may remain distinct and separate, thus presenting a much greater number of nuclei. The largest number of calculi found in any one case, according to Rolleston (1905), was recorded by Otto, who counted 7802.

It is probable that the stones usually originate in the gall-bladder, and that they are all formed nearly simultaneously; but when a solitary stone becomes impacted in one of the ducts, we see no good reason for disputing the possibility of the subsequent formation of other stones in the stagnated bile, provided the factors necessary for gall-stone formation are still present.

According to Rolleston, Mignot concluded from experimental researches that additional calculi might be formed in recurrent attacks of cholecystitis. Reference has already been made (p. 422) to the teaching of Aschoff and Bacmeister, that the "radial cholesterin stone" is always formed first, without bacterial infection, and that subsequent bacterial invasion causes the deposition of calcium bile-stones. Naunyn drew attention to the fact that where numerous calculi are found, they are almost always alike in general appearance, and came to the conclusion that gall-stones in a single individual always were formed from a single infection of the gall-bladder, and that recurrences of such infection in that particular individual were very rare. Kehr (1901) thought that the vast majority of stones are formed in the gall-bladder and that those instances of stone formation taking place within the ducts are "extremely rare."

Gall-stones may be **classified** in many ways. Most authors follow Naunyn, who described six varieties:

1. *Pure Cholesterin Stones.*—These are more or less uncommon, although this variety is the one that Aschoff and Bacmeister claim precedes the formation of all other varieties. As a rule they are of large size. They generally present the following characteristics: they are oval, at times being spheroid; hard, but brittle, so that they may be crushed between the thumb and finger; the surface may be either smooth or nodular; in color they vary from pure white or yellowish to brownish-black on the surface, with a white and generally crystalline interior. They are not stratified, the cholesterin crystals being radially arranged around a comparatively soft center of amorphous material (Figs. 133, 134). Rarely an amber like calculus may be found—amber yellow, translucent, irregular on the surface, and with the consistency of rosin.

2. *A Laminated Variety of Cholesterin Stones.*—These are composed of cholesterin, bilirubin-calcium, biliverdin-calcium, and calcium car-

bonate. These calculi usually contain about 90 per cent. of cholesterin. They may occur singly or in varying numbers, when they are at times faceted. A section reveals laminations, layers of almost pure white cholesterin alternating with yellowish, brown, red, or green layers. The body of the stone generally is *non-crystalline*, while the center or

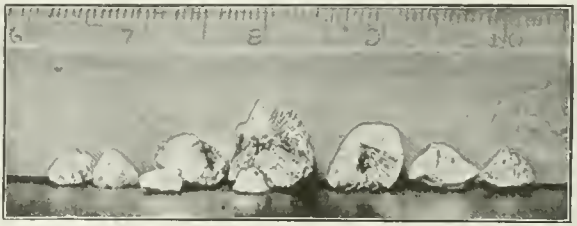


FIG. 133.—Radial Cholesterin Gall-stone; Spontaneous Fracture in Gall-bladder. Female, Aged 50 Years, with Empyema of Gall-bladder. Recovery. Scale in Inches. (See Fig. 134.) From a Patient under the Junior Author's Care in the Episcopal Hospital. (*Ashhurst's Surgery.*)

nucleus may be crystalline or may be hollow when the calculus has become dry. These calculi, according to Aschoff and Bacmeister, usually are formed by accretions around the primary "radial cholesterin stone" as a nucleus.

3. *The Common Gall-stones, or Mixed Cholesterin Calculi.*—They vary greatly in number and in size and usually are faceted (Plates V and VI). The surface varies in color from yellow, the usual color, to a brown or at times, a white color. Their consistency depends a great deal upon the time of their examination. When freshly removed from the gall-bladder they may be rather soft with a "soapy" feel; after they have dried out they become harder. The center of this variety is also, at times, a cavity; it usually is softer than the outside.

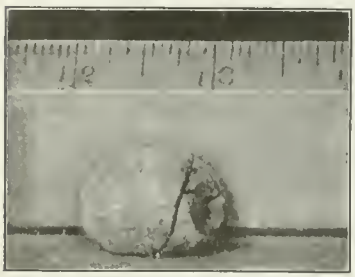


FIG. 134.—Cholesterin Gall-stone, with Polished Facet at Each End. Same Stone Shown in Fig. 133 after Fragments had been Glued Together. (*Ashhurst's Surgery.*)

4. *Mixed bilirubin-calcium stones*, in which there is a preponderance of the bile-pigment salt, with about 25 per cent. of cholesterin. They are often of considerable size. They generally occur singly or in groups of three or four, in which instances they may be faceted. On section they show concentric layers of dark material which is generally reddish-brown in color. The interior of the stones as a rule is rather soft; on drying, the stones usually contract with the formation of fissures.

Specimen Secured by Cholecystectomy, Showing Cholelithiasis with Patch of Gangrene and Threatening Perforation. *Lankenau Hospital.*

Face p. 426

5. *Pure bilirubin-calcium stones,* which are of two types: They may be small brownish-black calculi, with irregular surfaces, and showing a tendency to adhere one to the other; this type is rather soft in consistency. The other type is much larger and much smoother on the surface, which usually exhibits a metallic luster, while the interior of the stone is rather spongy in consistency.

6. In this class Naunyn places some *rare forms* including among them calcareous stones, pearly bodies which consist of amorphous or partly crystalline cholesterin, casts of the biliary passages, etc.

The pathology, symptomatology, diagnosis, and treatment of gall-stone disease will be considered in a subsequent chapter (see page 460).

Typhoid Infections of the Biliary Tract

The infections of the biliary tract by the typhoid and para-typhoid bacilli are of great importance. Not only because of the surgical conditions that arise from such infections is this true, but also from the standpoint of public health and hygiene. Many epidemics of enteric fever have been traced directly to persons who were suffering from a more or less remote typhoid infection of the gall-bladder and bile-ducts. The usual medical measures, such as cholagogues, are of no value in ridding the biliary tract of the Bacillus typhosus. Nichols (1917), however, on the theory that increasing the alkalinity of the bile tends to sterilize it, treated two patients successfully by administering daily, for about ten days, two grains of sodium carbonate. The efficiency of the vaccine treatment has not been established; though Dixon (1917) employed it successfully in one patient, in the case reported by Leary (1913) it was entirely useless. It has thus become necessary for the surgeon to devise ways and means for accomplishing the end in view—ridding the system of the remaining typhoid bacilli. The treatment of these patients is considered at page 433.

Infections of the gall-bladder and bile-tracts have been recognized as complications of typhoid fever by almost every one who has studied many cases of the latter affection. Since 1897, when Mason reviewed the history of this complication of typhoid, numerous investigators have carefully studied the subject and, as Kelly says, have reached a comparatively unanimous conclusion "(1) that the typhoid bacillus is regularly present in the gall-bladder, and commonly in pure culture, in practically all cases of typhoid fever—indeed, it is the one region of the body from which a pure culture of the organism is most likely to be obtained; (2) that the typhoid bacillus may persist in the gall-bladder,

as well as within gall-stones, weeks, months, even years after the patient has recovered from an attack of typhoid fever; (3) that cholangitis and cholecystitis (catarrhal, suppurative, and gangrenous) are by no means infrequent complications of typhoid fever; and (4) that a history of antecedent typhoid fever may be obtained in many cholelithic and cholecystic subjects."

The presence of the bacilli in the gall-bladder is explained in two ways. It is the generally accepted theory, as explained at page 417, that bacteria are constantly passing from the intestinal tract to the liver, in the portal circulation. Normally these bacteria are destroyed in the liver; but if the liver cells become overburdened by the work thrown upon them, or are poisoned by the toxins thrown out by the bacilli, their bactercidal power is lost, and live bacilli are then excreted with the bile. That infection may occur also by the systemic circulation is suggested by the observations of Joseph Koch, Wrzosek, and Else. Koch discovered inflammatory infiltration in the mucous membrane in a case of beginning typhoid cholecystitis, the infiltrate containing dense clumps of bacilli resembling capillary emboli. From these foci, he claims, the bacilli pass through the epithelium into the interior of the gall-bladder. In experimental work on rabbits he was able to follow the transit of the bacilli from the capillary emboli in the submucosa through the walls of the gall-bladder. In these experiments he injected typhoid bacilli into the veins of rabbits and recovered them from the gall-bladder even in those cases in which he had previously tied the cystic duct.

Although the excretion (or secretion) of bile by the liver cells is almost constant, its discharge into the duodenum varies with the needs of the digestive functions. During the periods of digestive quiescence the bile backs up through the cystic duct into the gall-bladder where it remains until needed in the intestinal tract. Typhoid fever patients are kept on an extremely low diet, large quantities of bile not being required for its digestion and absorption. In consequence of this fact, the gall-bladder naturally becomes distended with bile, and the latter becomes more or less loaded with mucus. If the mucus causes a partial obstruction of the cystic duct, the bile will become stagnated, allowing rapid multiplication of any bacteria that may be present. Under these conditions, with good resistance on the part of the patient, there will result a slight inflammation in the gall-bladder,which is overlooked in many instances because the patient's sensibilities are numbed by the typhoid infection and he does not experience sufficient pain to attract the attention of the physician; and the physician will overlook

it, unless, as Kelly urges, "systematic and repeated examinations of the gall-bladder region are undertaken."

With lessened resistance, the bacilli are allowed greater freedom of action, and there results a more pronounced cholecystitis, with or without ulceration of the mucous membrane or even of the entire wall of the gall-bladder, and, at times, perforation of that viscus.

In the presence of pre-existing cholelithiasis, infection of the gall-bladder during the course of typhoid fever might be expected to cause marked inflammatory changes, due to the fact that pathological lesions already exist in that viscus as a result of the presence of the calculi.

No conclusive statistics as to the frequency with which the gall-bladder is involved in typhoid fever have been published. The junior author in analyzing (1908) the reports of the Episcopal Hospital found that among 2864 cases of typhoid fever there were but eighteen or 0.62 per cent. in which infection of the gall-bladder was recorded as a complication. Thomas (1907), in a series of 895 cases of typhoid, found cholecystitis in twelve or 1.3 per cent.

In connection with two cases of perforation of the gall-bladder during typhoid fever treated by operation, the junior author analyzed the reports of nineteen other operations on the gall-bladder during typhoid fever.[1] The lesions found at operation were as follows:

Operations for gall-bladder lesions during typhoid fever	Cases	Recovered	Died	Mortality per cent.
Cholecystitis alone	4	2	2	50.00
Cholecystitis and empyema of the gall-bladder	3	0	3	100.00
Cholecystitis, empyema, peritonitis	4	2	2	50.00
Perforation with peritonitis	6	4	2	33.30
Perforation (found only at autopsy)	4	0	4	100.00
Total	21	8	13	61.90

Quénu (1908) collected forty-five operations for lesions of the gall-bladder during or soon after an attack of typhoid fever, and Reid and Montgomery (1920) have collected 18 cases occurring in patients less than 15 years of age.

Thomas has analyzed 154 cases of typhoidal cholecystitis collected from the literature. He reports that perforation of the gall-bladder occured in thirty-nine or 25.3 per cent. Eleven of these patients were operated upon with a mortality of 54.6 per cent. The remaining twenty-eight died without operation. The Bacillus typhosus was

[1] Price (1916) collected nine further such operations, with one death.

isolated in about 50 per cent. of the cases subjected to operation; calculi were found in three. In the series reported by the junior author the typhoid bacillus was recovered in pure culture from the gall-bladders of nine patients or 42.8 per cent. In one case, the typhoid bacillus was associated with the colon bacillus; in another case the paracolon bacillus was found; in ten cases the bacteriological findings were not recorded.

Symptoms and Treatment of Gall-bladder Disease during Typhoid fever.—There are two quite distinct classes of cases: In the first there is a more or less gradual onset of abdominal pain, fairly well localized (by patients who are conscious) to the right hypochondrium, accompanied by localized tenderness, and frequently by a palpable

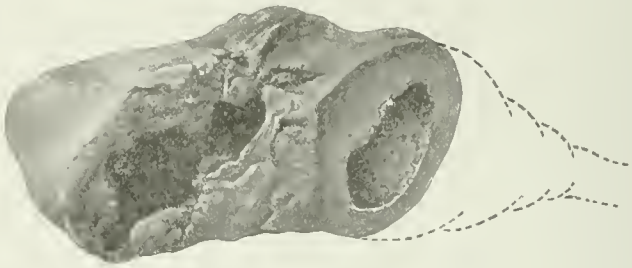


FIG. 135.—Perforation of the Gall-bladder during Typhoid Fever; Cholecystectomy; Recovery. Natural Size. From a Patient under the Junior Author's Care in the Episcopal Hospital.

mass easily recognized as the distended gall-bladder. If operation is undertaken at this stage, there is found (*a*) cholecystitis; (*b*) empyema; or (*c*) empyema and commencing suppurative pericholecystitis. In the second class of cases the symptoms already mentioned have existed for a period varying from a few hours to a week or ten days, when suddenly there is an acute attack of abdominal pain, accompanied by a fall of temperature (noted in eight out of ten cases), and sometimes by sweating; these signs being gradually followed, when immediate operation is not undertaken, by a secondary rise of temperature, a spread of the pain and tenderness over the whole abdomen, and increase of distention as the initial rigidity disappears.

When cholecystitis is recognized as a complication during an attack of typhoid fever, it is not desirable to resort immediately to operation. The condition should be closely watched, and only when it is thought that empyema is present or perforation threatens, should the gall-bladder be drained. If perforation occurs, no delay should be allowed; in most cases cholecystectomy is the operation of choice. If the cholecystitis subsides without operation, the patient should be kept

under observation, and such dietetic and hygienic treatment should be instituted as is suitable in cases of cholelithiasis, since the subequent development of calculi is very probable.

Sequels of Typhoid Infection of the Biliary Tract.—It is almost impossible to arrive at definite conclusions in regard to the sequels of typhoid infections of the biliary tract. In a series of 521 operations for cholelithiasis performed by the senior author at the Lankenau (formerly German) Hospital a positive history of antecedent typhoid was obtained from 127 patients or 24.3 per cent. The time elapsing between the attacks of typhoid and the time of operation varied from two weeks to forty-one years. It is questionable whether all of these cases should be classed as sequels of typhoid fever, and there is no way of deciding this question. A decision based solely on the bacteriological findings leads to one conclusion; but if based on the accepted pathogenesis of gall-bladder infections (recollecting that the gall-bladder may rid itself of all bacteria after the subsidence of the acute infection) a different conclusion may be reached. Among a series of eighty-one patients, at the Lankenau Hospital, with a history of antecedent typhoid infection, the contents of the gall-bladder were examined bacteriologically in forty-six; of these, thirteen cases, or 28.4 per cent., gave cultures of the bacillus typhosus. It cannot, however, be stated positively that none of the remaining sixty-eight cases should be classed as sequels of typhoid fever; since these might be cases in which the gall-bladder had rid itself of the Bacillus typhosus after this infection had caused structural changes, the deposition of concretions, etc. In fifteen or 32.6 per cent. of the forty-six examinations, pure cultures of the colon bacillus were obtained. Here again it is uncertain whether these cases should be classed as sequels of the antecedent typhoid, or as cholelithiasis resulting from the invasion of the gall-bladder by the colon bacillus; but we believe that the calculous formation in these cases, with possibly a few exceptions, was a direct result of the pathological changes consequent upon the typhoid infection. There might also be classed in the same category seventeen (36.9 per cent.) of the forty-six cases in which the gall-bladder was sterile at the time of operation, and one case in which Staphylococcus aureus was found.

A general summary of the thirteen cases giving a pure culture of the Bacillus typhosus follows:

Sex.—All were females. *Age.*—The age varied from twenty-three to fifty-six years, at time of operation, the average age of the thirteen being 40.3 years. *Time since convalescence from typhoid:* This varied from two weeks to forty-one years, the time being respectively,

two weeks; one month; five weeks; four months; five months; nine months; three years; seven years; eight years; fourteen years; fifteen years; forty-one years; one case not stated. *Operations:* Cholecystectomy was performed in six cases; cholecystectomy with choledochostomy, in three; cholecystostomy in three; choledochostomy in one. *Results:* All recovered.

Thomas in his analysis of 154 cases, quoted above, which he classed as complications or sequels of typhoid, found the Bacillus typhosus in 50 per cent.; but in those patients in whom the gall-bladder condition arose during the course of typhoid fever, the typhoid bacillus was present in the gall-bladder in perhaps 95 per cent.

More definite findings will have to be obtained before cases similar to those in the various series mentioned above can be classed with certainty as complications or sequels of typhoid, and not acute or chronic diseases of the biliary tract in patients in whom an antecedent attack of typhoid fever was but a coincidence, although we believe that most of these cases should be placed in the former category; certainly when the typhoid bacillus is found in the gall-bladder the presumption is warranted that it has caused the disease.

Another very interesting condition has been made evident by bacteriological examination made as routine in cases of operation on the gall-bladder. This is the fact that *infection of the biliary tract by the Bacillus typhosus may occur in patients who have never presented any symptoms of typhoid fever,* the invasion giving rise to a primary typhoid cholecystitis. Quénu (1908) pointed out that typhoid fever is not primarily an intestinal disease (wherefore the term *enteric* fever is to be avoided), but a primary septicemia; and that the liver is the primary agent for the elimination of the Bacillus typhosus, which is brought to it by the blood. In this manner the bile becomes infected; and he asserts that many jaundices—hepatic, epidemic, or simple febrile in character—are nothing less than primary infections of the bile passages by the bacillus typhosus.

Typhoid Carriers.—From the standpoint of public health, the most important cases of typhoid infections of the biliary tract are found in those individuals who are chronic "*typhoid carriers.*" In this condition the patient recovers from the attack of typhoid but does does not rid himself of the typhoid bacilli, which remain, usually in the gall-bladder, being discharged from that viscus into the intestinal tract and excreted with the feces. Some of these persons give no history of an illness resembling typhoid fever. Repeated instances of epidemics arising from such sources have been brought to the attention

of the medical profession, infection generally occurring through the milk supply or through food prepared by such a typhoid carrier. The proportion of chronic carriers has been estimated at from 1.7 per cent. to as high as 6 per cent. of the total number of typhoid cases (Garbat, 1916). The bacilli have been found in the livers of these patients at autopsy, as well as in the walls of the gall-bladder and in the bile (Hammond, 1909). The typhoid bacilli may remain in the gall-bladder for an indefinite period. Gregg, according to Kelly, recovered the bacillus fifty-two years after the subsidence of the acute attack; and Jundell (1908) reported twenty-two cases of infection extending over a period of fifty-four years, arising from a "carrier" who herself had never had an attack of typhoid fever.

The diagnosis of "chronic carrier" is not made from any symptoms referable to the gall-bladder or gall-ducts. Usually the patients are perfectly normal symptomatically, so far as the liver and biliary tract are concerned. The only positive means of diagnosis is finding the bacilli in the stools of the suspected "carrier," or in the duodenal contents. The latter is the more reliable method if the bacilli have their habitat in the biliary tract. It must be remembered, however, that cases have been reported in which the intestinal tract appeared to harbor the infection, the bile itself being normal.

In spite of the occasional success of non-operative treatment, as mentioned at page 427, we believe that operation is indicated in the majority of typhoid carriers. The operation should consist in cholecystectomy and drainage of the hepatic duct as advocated by Kehr (1913); if after operation repeated tests show the bile coming from the liver to be sterile, it may be safely assumed that the gall-bladder contained the infecting focus; if the bile remains infected, only prolonged external drainage, as in other cases of cholangeitis, will procure its sterilization. In the latter cases vaccine or other medical treatment may prove efficient after external drainage of the bile even if employed without success previous to operation. For the rare instances of *intestinal* typhoid carriers, operative treatment so far as we are aware, has not been adopted; but it is logical to suppose that drainage of the tower ileum (*ileostomy*) or cecum (*cecostomy*) might be curative. The treatment of typhoid carriers whose urinary tract is infected, and in whom the kidney is the focus, does not come within the scope of this work.

Few surgeons have had opportunity to operate on more than one or at most two typhoid carriers, and results in isolated case reports are apt to be misleading. Nichols, Simmons and Stimmel (1919), how-

ever, report a consecutive series of six chronic typhoid carriers from the Walter Reed General Hospital: one (a "urinary" carrier) was cured by nephrectomy, three were cured by cholecystectomy, and two failed of cure by cholecystectomy. It is to be noted, however, that the importance of hepatic duct drainage was not generally recognized at the time these operations were done; better results may be anticipated in the future.

Jaundice as a Symptom

Jaundice as a sign or symptom is always of importance to the surgeon. The term signifies a peculiar discoloration of the tissues by bile-pigment. It is not a pathological entity, but a symptom of a number of lesions that are found in connection with diseases of the liver and bile-passages, or the structures adjacent to them.

In a healthy individual, bile-pigment is found only in the bile. Under normal conditions these pigments are formed in the liver; and the liver or its excretory ducts are involved clinically, with very rare, if any, excpetions, in every case of jaundice. It is essential for the diagnostician to differentiate between those cases requiring surgical treatment and those that will be relieved by medical measures alone.

In the majority of cases jaundice results from absorption of bile from the small radicles of the bile-ducts, due to obstruction of these channels. When it is realized that the secretion-pressure of the bile amounts to only 200 mm. of water, it is evident that even slight obstruction to the flow of bile will overcome its secretion-pressure. When obstruction exists in the bile-passages, either intrahepatic or extrahepatic, there is a damming up of the bile in the proximal portions of the ducts extending as far as the capillary radicles. As the secretion of bile continues, increased dilatation of the ducts follow. The capillaries gradually become lengthened and distended until they rupture into the adjoining lymph-spaces. The bile-pigments are then absorbed by the lymphatics, and are carried to the thoracic duct and into the general circulation. That the bile-pigments are carried to the blood through the thoracic duct has been repeatedly demonstrated (Kelly, 1908). After the common bile-duct is ligated bile-pigments are soon recovered through a fistula in the thoracic duct; when both thoracic and common ducts are ligated, thus preventing the adminsion of bile into the general circulation through the thoracic duct, discoloration of the skin is seldom seen, and the bile-pigments do not appear in the urine for several days. It is also possible for the bile-pigments to

enter the systemic circulation through the intrahepatic capillaries (Quincke, 1903).

There are numerous instances of icterus occurring in patients where it is impossible to demonstrate an obstruction to the bile-ducts. In such cases the manner of absorption of the bile-pigments is not so plain. But it is probable that the mechanism is much as described above, although various theories have been advanced to account for the occurrence of jaundice when no obstruction is evident. According to Kelly, Eppinger believed that obstruction takes place even in these instances, but only in the most minute radicles of the bile-channels: stasis of bile occurs and there is dilatation of the biliary canaliculi with final rupture into the pericellular lymph-spaces.

In some instances of injury to the liver or gall-ducts with escape of free bile into the peritoneal cavity, jaundice follows its absorption. If there is a chronic peritonitis present, however, the peritoneum will be incapable of absorbing the bile-pigments, and jaundice may not occur.

The presence of bile-pigments in the circulating blood gives rise to certain **symptoms** which are entirely independent of the primary cause of the condition. These are most marked in obstructive jaundice, and vary somewhat in degree with the completeness of the obstruction. The most noticeable symptom produced is discoloration of the skin and visible mucous membranes. In the onset of icterus the color is generally a light yellow, gradually changing to a greenish tint, and terminating in a yellowish-gray. The natural pigmentation of the skin will often mask the lighter degrees of jaundice, so that it is often overlooked in brunettes. The scleræ, as a rule, are discolored more rapidly than the skin. In many patients a *subicteroid* tint of the skin is an evidence of a mild chronic cholangeitis; such patients may never have had a true jaundice and it may have been thought they were merely swarthy.

Itching of the skin is more or less prominent as a symptom of icterus, often preceding the discoloration, although as a rule it follows the latter condition. There is no relation between the intensity of the discoloration of the skin and the degree of pruritus.

It is said that xanthoma (xanthelasma) may occur in chronic lithiasis with jaundice. This skin lesion is not peculiar to jaundice although often seen in connection with it. It consists of pale yellowish spots, found chiefly in the region of the eyelids, the areas of discoloration being raised above the level of the surrounding skin. According to Quincke the yellowish pigmentation of these spots is not caused by

bile-pigments, nor has their connection with icterus been definitely determined.

The discoloration of jaundice is frequently observed first on the visible mucous membranes, especially the scleral conjunctivæ. This change is not always apparent on the other mucous membranes except on the lips and hard palate, where the membrane is naturally more or less pale. It usually can be demonstrated on the visible mucous membranes by depriving them of their blood by pressure, as by that of a thin glass slide against the everted lower lip. But the discoloration of jaundice may often be apparent on the abdomen when not noticeable in the conjunctivæ.

The excretion of the greater part of the bile-pigments through the kidneys results in discoloration of the urine, the color varying from yellowish, through yellowish-red, to a greenish-brown depending to a great extent upon the presence of the different pigments and their products. This discoloration of the urine is generally seen before the pigmentation of the skin, preceding the latter at times by several days, being in reality the first clinical symptom of jaundice. It can be demonstrated by the discoloration of the froth after a brisk shaking of the urine; and often by the staining of the filter-paper through which the urine is passed. The more delicate tests are given in text-books of medicine and in laboratory manuals.

In protracted cases of jaundice, the urine may be diminished in amount, with increase in the specific gravity. Hyaline casts, at times bile-stained, are found.

The pale "acholic" stools of a jaundiced patient are another diagnostic sign (p. 437).

Hemorrhage in Cases of Jaundice. Bile-pigments are present in the blood in every case of jaundice but their action on the blood has not been definitely determined. Jaundice, especially if of long duration, is attended by an impoverished condition of the blood with a diminution in the number of erythrocytes and in the amount of hemoglobin. There is also present a decided prolongation in the coagulation time of the blood. This will be increased from the normal, three or four minutes, to as much as fifteen minutes and even longer. This condition may depend upon a change in the fibrin ferment, upon alterations that occur in the red blood-cells, or may be due to the mere presence of the bile-pigments and bile-acids. When this condition is present there may be bleeding from the mouth, the nose, the intestines, the bronchi, the kidneys, and into the skin. After operation there is sometimes an oozing from the wound which will continue until the patient is exsan-

guinated. The oozing may be absolutely uncontrollable. This bleeding may not begin at the time of the incision; the wound may be dry when closed but two, three or even four days after the operation the dressings may be found soaked with blood, by an uncontrollable oozing that sometimes continues until the death of the patient. This danger cannot be prevented, but perhaps it may be lessened by the administration of calcium chloride in 2-gramme doses every three or four hours for a day or more before the operation, by mouth, and 4-gramme doses after the operation, by rectum. Calcium chloride reduces the coagulation time of the blood but it does not always suffice to prevent the oozing. Transfusion of blood, a remedy which is available in all well equipped hospitals, is the most certain remedy known. When this method cannot be employed, some form of ready prepared alien serum should be injected hypodermically or intravenously. Anti-diphtheritic serum usually may be procured, and should be used in doses of from 5000 to 10,000 units, or horse serum may be administered two or three times daily in doses of 10 to 30 cc. for several days after operation.

The **absence of bile-pigments from the stools** will cause the excreta to be lighter in color than normal; their complete absence produces the well-known "clay-colored stools." Variations in the color of the stools, showing alternately the presence and absence of bile-pigments indicates an intermittent obstruction to the flow of bile, a condition that is found in the so-called "ball-valve" obstruction of the common duct by a calculus. When obstruction is due to a tumor, such as cancer of the head of the pancreas, the obstruction does not vary; the stools remain constantly free from bile-pigments, and are of a grayish color.

The color of the stools seen in jaundice is partly due to an increase of the undigested fats which may increase from normal (7 to 10 per cent.) to as high as 80 per cent.

To comprehend the true significance of jaundice as a symptom in surgical diseases of the liver and biliary passages, we must consider the various causes which will give rise to it, and the frequency with which it is found in those diseases which are treated by the surgeon. Icterus occurs whenever there is a more or less prolonged obstruction of the hepatic or common duct, from any cause. It occurs, according to Kelly "under other circumstances—in which apparently the biliary ducts are patent, as in cirrhosis and other diffuse diseases of the liver; in many infections, such as the different types of so-called infectious jaundice, syphilis, yellow fever, septicopyemia, malaria, pneumonia, typhoid fever, etc.; in intoxications such as poisoning with ptomains, phosphorus, arseniuretted hydrogen, chloroform, mushrooms, toluyl-

enediamin, pyrogallol, snake venom, coal-tar products, etc.; in acute yellow atrophy of the liver; in progressive pernicious anemia and hemoglobinemia; in disturbances of the circulation, such as passive congestion; in certain nervous perturbations (so-called emotional jaundice, menstrual jaundice, etc.); in the new-born, etc."

Hunter (1897) gave the following classification of jaundice not due to obstruction:

1. Jaundice produced by the action of poisons, such as phosphorus, arsenic, and snake venom.
2. Jaundice met with in various specific fevers and conditions, such as yellow fever, malaria (remittent and intermittent), pyemia, relapsing fever, typhus, typhoid fever, and scarlatina.
3. Jaundice met with in various conditions of unknown or more or less obscure, infectious nature, and variously designated as "epidemic," "infectious," or "malignant" jaundice, "icterus gravis," "Weil's disease," and "acute yellow atrophy of the liver."

To these might be added the jaundice frequently associated with severe hemorrhage, with starvation, or with lowered blood-pressure in the portal or hepatic vessels in the presence of increased tension in the smaller bile-ducts.

The following is a convenient classification of jaundice due to obstruction:

1. Obstruction due to inflammatory thickening of the mucous membrane of the ducts, or the result of such inflammation; rarely to tumors of the ducts themselves.
2. Obstruction due to stone in the common, rarely the hepatic, duct; or in the cystic, pressing on the common duct.
3. Obstruction due to neoplastic or hyperplastic conditions of the pancreas, or its lymphatics, especially those of the head of this organ.
4. Obstruction due to neoplasms or to pathological conditions such as adhesions, enlarged lymph-nodes, kinks, etc., of neighboring organs exerting pressure on the common duct.

Jaundice does not occur in every case of biliary tract disease, but a carefully taken history will show that it has been present at some period of the disease in about 65 per cent. of the cases, and has not

been noted in about 35 per cent. At the time of operation, in our own experience, jaundice is present only in about one-third of the patients. Mayo, however, has stated that jaundice was present in 70 per cent. of his patients at the time of operation.

Jaundice is a symptom and not a disease; in every case there is a lesion to account for the presence of this symptom, but great difficulty is often encountered in determining just what this lesion is. A complete history and careful study of the accompanying symptoms will, in the majority of cases, make the diagnosis clear; in other cases an exploratory operation will be necessary.

The following aphorisms may be of assistance in determining the cause of jaundice:

Jaundice which is slight and persists most probably is independent of obstruction.

Jaundice from obstruction becomes intense very rapidly.

Jaundice coming on gradually but ultimately becoming intense, with clay-colored stools, generally is due to pressure from neighboring structures, especially to diseases of the pancreas, such as pancreatic lymphangeitis, chronic interstitial pancreatitis, or carcinoma of the pancreas.

Jaundice which does not persist indefinitely or which recurs time and again, generally is due to calculous obstruction.

Jaundice with sudden onset, accompanied by colicky pains and clay-colored stools, generally is due to obstruction within the gall-ducts.

Jaundice following severe paroxysms of pain generally is due to gall-stone formation or to carcinoma. In the latter case there should be a history of failing health before the onset of pain or jaundice.

Jaundice in the presence of an enlarged liver generally is due to cirrhosis of the liver, to cancer of the liver, or to pyemic abscess of the liver.

Jaundice with ascites generally is due to cancer of the liver or to cirrhosis. In the former there are darting pains, loss of weight and intense jaundice. In cirrhosis there generally is a history of alcoholic dyspepsia; the jaundice is generally much less intense than in cancer.

Jaundice with pyrexia is secondary either to acute febrile infection, to suppurative pylephlebitis, or to inflammation of the bile-ducts. Temporary pyrexia may be caused by the passage of a stone through the bile-ducts.

Jaundice with a history of previous attacks generally is due to a catarrhal condition of the bile-ducts or to the presence of gall-stones.

Jaundice with cerebral symptoms generally is due to acute atrophy of the liver, to poisoning by phosphorus, or to some specific fever such as pneumonia.

Jaundice in a young person preceded by symptoms of gastric catarrh generally is "catarrhal jaundice" (page 445).

Jaundice which is intermittent, at times slight and again intense, with urine that varies in color from light to dark, with stools that are intermittently dark and clay-colored, with colicky pains, with chills and fever similar to those of malaria, almost invariably is due to chronic calculous obstruction of the common duct.

Jaundice with fatty stools, in the presence of glycosuria, is generally indicative of pancreatic disease.

Jaundice increasing, without remissions, with marked dyspeptic symptoms, with marked increase in neutral fats in the stools, with undigested muscle fibres with nuclei intact in the stools, is due to pancreatic disease.

Jaundice following general failure of health, increasing until it becomes absolute and never varying, with a greenish tint, with rapid loss of weight, with a distended gall-bladder, with gradual and painless onset, generally is due to carcinoma of the head of the pancreas.

In jaundice due to obstruction, either internal or external, the stools are clay-colored or lighter in color than normal, either continuously or intermittently.

In jaundice not due to obstruction of the bile-ducts, the stools are not clay-colored. It must be remembered that certain drugs, such as bismuth, iron, and charcoal will color the stools; altered, disintegrated blood in the stools will also change the color.

Jaundice is seen in non-calculous cholecystitis only after the inflammatory process in the gall-bladder has extended to the mucous membrane of the cystic and common ducts.

The significance of jaundice as a symptom in various diseases of the liver and biliary tract will be considered more fully in the sections devoted to those lesions.

CHAPTER XVII

SURGERY OF THE GALL-BLADDER AND BILE-DUCTS

Deformities, Anomalies and Malpositions

Congenital Obliteration of the Bile-ducts.—The absence of a passageway for the bile, at birth, usually is the result of obliteration or atresia of the common bile-duct. Lavenson (1908) suggested that the condition is a true atresia, and not an "obliteration," the latter presupposing the existence of a lumen which subsequently becomes destroyed. The condition, although unusual, is not rare. Holmes (1916) collected 108 authentic cases from the literature.

Paul Mathieu (1908) collected eight authentic and twelve doubtful cases of **congenital stricture** of the bile-ducts; the stricture usually has been at or near the duodenal termination of the common duct.

Other rare anomalies include cases where there is more than one common duct, cases where one or more of the bile-ducts open into the stomach or at an unusual point in the intestine, and anomalies in the number and situation of the hepatic ducts.

Pathologically, there is an obstruction, atresia, or obliteration of the common duct, with cirrhosis of the liver. Lavenson thought the cirrhosis is the result of the obliteration and consequent bile stasis; Rolleston (1907) supported the view that the cirrhosis precedes the obstruction, a descending cholangitis causing obliteration of the lumen of the duct. As pointed out by Rolleston, other causes of stricture must be recognized, such as fetal peritonitis, with its resulting adhesions; or obstruction of the ducts by syphilitic granulation tissue, as in a case under his own care.

The classification of these cases of infantile jaundice which has been adopted by Mathieu is convenient. He classifies them in two main divisions: (1) Those in which the condition is not compatible with life (congenital occlusion or total absence of the bile-duct); and (2) cases compatible with life, including both (*a*) congenital icterus without lesion of the bile-ducts (which may be attributed to angiocholitis of the intrahepatic ducts or to splenic disease causing jaundice by hemolysis), and (*b*) those cases already mentioned, in which stricture but no absolute occlusion of the ducts exists.

Clinically congenital obstruction of the common duct is characterized by jaundice which generally is present at birth, although it may not develop for some weeks. The jaundice increases in intensity, the urine contains bile and the stools are free from all bile-coloring matter. The liver and spleen generally are enlarged. Hemorrhage from the cord may occur, and purpuric spots may be seen. Emaciation rapidly ensues, with coma, stupor, and, at times, convulsions followed by death. One case reported (Treves) where the patient lived nineteen years with continuous jaundice for sixteen years.

The *diagnosis* is not readily made. The condition may be distinguished from icterus neonatorum by the short duration of the latter, and the presence in that disease of bile in the stools and its absence from the urine (Griffith, 1908). In the jaundice of the newborn due to a simple catarrhal duodenitis or cholangitis, there always is some degree of fever, which usually is absent in cases of icterus due to congenital anomaly or stricture. Partial occlusion of the duct causing jaundice which ultimately will clear up cannot be distinguished until time has proved the diagnosis. A duodenal catheter is an aid in diagnosis: being passed through the stomach and pylorus, the duodenal contents are pumped out, and are examined for bile, pancreatic juice, etc. In Hess's patient life was preserved for three months, possibly because the accessory duct of the pancreas was patulous. Though tests made during life showed the presence of bile in the duodenal contents, complete occlusion of the common duct was found at autopsy; and Hess suggests that perhaps bile may be excreted from the circulation through the intestinal canal.

The *treatment* is operative, although antisyphilitic treatment should be instituted in the hope that the condition is due to congenital syphilis, even if the history of the patient is negative. The surgical procedure necessary will be some form of anastomosis between the gall-bladder or the upper, unobliterated portion of the duct, and the duodenum (page 510). Lavenson collected reports of four unsuccessful attempts at operative relief.

Congenital Absence of the Gall-bladder.—Although the gall-bladder is not essential to life and is entirely absent in some of the lower animals, such as the rhinoceros, the camel, the goat, the deer and the elephant, its congenital absence in man is rarely noted. Nearly all of the important information upon the subject, as pointed out by Stone (1908), has been assembled by F. Fink (1904) and A. Bubenhofer (1905). In most of the cases reported as such, as pointed

out by Eshner (1894), "the absence of the gall-bladder was associated with conditions that point to obliteration of a previously existing viscus, rather than to a condition of agenesis." Eshner reported the case of a child who came under his observation for a persistent cough. The bowels moved several times daily, the stools being pale. No jaundice was present. The patient died when two years of age, and at autopsy the liver was found to be of normal size and condition, although changes probably syphilitic in origin were apparent. The biliary vessels and ducts, with the exception of the cystic duct, were normal. The gall-bladder was absent, the usual fissure for the gall-bladder was wanting, and there was nothing suggestive of the gall-bladder ever having been present. Gay (1902) collected nineteen cases of absence of the gall-bladder; in six of them "absence" was the only abnormality. Stone's patient was a woman fifty-four years of age upon whom an operation was performed under the diagnosis of calculous cholecystitis, with obstruction of the common duct. A palpable mass was thought to be the gall-bladder filled with calculi. Upon opening the abdomen the gall-bladder was conspicuously absent, the duodenum and pylorus being attached to the normal site of that viscus. No adhesions were present. Calculi were readily palpated in the common and hepatic ducts. After the ducts had been opened and the stones removed, search for the gall-bladder was made but it could not be found. Exploration with a probe through the hepatic duct failed to reveal any vestige of the cystic duct. Niemack (1908) has reported a case of cholesterin stone in the common duct in a patient in whom he could not find a gall-bladder.

The condition does not present any sign or symptoms that might lead to a correct diagnosis. The diagnosis is made either at the operating-table or postmortem. The *treatment* of the cases subjected to operation should be directed to the condition for which the operation is performed.

Anomalies in position and shape of the gall-bladder are not common. Constrictions, sacculations and other deviations from the normal shape of the viscus may be the result of inflammatory changes; the organ may become enormously distended as a result of obstruction of the cystic duct or may become practically obliterated by cicatricial contraction. Many of the cases of "hour-glass" gall-bladders that have been reported may be the result of a defect in development or the effect of cicatricial contraction. The gall-bladder may be bi-lobed, may be transversely placed, may be imbedded in the liver substance, or may be freely movable owing to the presence of a distinct mesentery.

This condition is described as **wandering gall-bladder,** and is not exceptionally rare. Kübig (1912) noted having seen two cases at autopsy. Its importance arises from the fact that it predisposes to torsion or volvulus of the gall-bladder.

Sherren (1911), who successfully removed at operation a **double gall-bladder,** refers to the autopsy reported by Purser (1886) as the only case where there existed two complete gall-bladders with separate cystic ducts; Purser himself quotes from the Philosophical Transactions (1693-94) a case where at postmortem there were found two distinct gall-bladders, one in the right lobe and the other in the left lobe of the liver. Schachner (1917) collected five cases, including his own patient in whom he drained both gall-bladders with satisfactory result.

FIG. 136.—Hour-Glass Gall-Bladder. From a Patient in the Lankenau (formerly German) Hospital.

Several cases of **hour-glass gall-bladder** are on record. The following case was observed at the Lankenau Hospital.

Hour-glass Gall-bladder.—A. S., female, aged thirty five; operation, May 24, 1904. Adhesions between the gall-bladder and stomach. These were ligated and cut, and the gall-bladder was found to be "hour-glass" in shape, both pouches being filled with calculi (Fig. 136). Cholecystectomy was performed, the patient recovering.

CHOLANGEITIS AND CHOLECYSTITIS

A thorough knowledge of the etiology and pathology of the infectious diseases of the biliary tract is essential for the correct interpretation of their signs and symptoms. These vary greatly with the different pathological processes, and an accurate diagnosis can be made, the proper treatment instituted, and the ultimate outcome predicted, only through a correct interpretation of their significance. In some instances the lesions are varied and multiple, being diagnosticated correctly only after direct inspection and palpation. In the majorty of cases, however, a study of the signs and symptoms present will enable the surgeon to make a correct diagnosis.

All non-neoplastic surgical diseases of the biliary tract are the result of bacterial infection.[1] Infection of the biliary tract gives rise to in-

[1] For a full description of the bacteria found in the biliary tract and their paths of entrance, see page 415.

flammatory changes in its component parts, the mildness or severity of the lesion produced depending upon the virulence of the bacteria and the resistance of the patient. Mild or severe cholangeitis, mild or severe cholecystitis, with or without the formation of biliary calculi, will follow. At times the infection is so slight and so readily thrown off by the involved structures that no permanent pathological lesions result. In other instances the onset and course of the infection are very insidious, the resulting chronic inflammation, gall-stone formation, etc., not giving rise to important symptoms until many years after the true commencement of the disease.

Infections of the biliary ducts and gall-bladder usually coincide. Infection of the gall-bladder may precede that of the ducts, but usually the ducts are first affected; the infection in the ducts may subside while that in the gall-bladder continues; but infection of one without infection of the other is rare. The results of the infection, however, vary greatly: thus pathological lesions which result from infection of the gall-bladder without serious, or with quickly subsiding infection of the bile-ducts, are very different from those seen when the bile-channels are permanently diseased. With involvement of the common and hepatic ducts, lesions of varying degrees of severity are always found in the liver; when the gall-bladder alone is infected, the liver is seldom involved. Infection of the ducts gives rise to hepatic and perihepatic lesions; infection of the gall-bladder without involvement of the ducts, gives rise to pericholecystic lesions.

Cholangeitis.—The simplest form of infection of the bile-ducts causes **acute catarrhal cholangeitis.** Usually this has the same etiology as gastro-intestinal catarrh, which often is described by its most striking symptom, as "catarrhal jaundice." Cholangeitis frequently is a complication or sequel of this condition. It is also observed in connection with some of the infectious diseases, such as typhoid fever and pneumonia; it may result from interference, by various toxins, with the normal activity of the ultimate radicles of the ducts in the liver, or with the metabolism of the liver cells; it may occur in cirrhosis of the liver, or in advanced heart disease.

Catarrhal jaundice is essentially obstructive jaundice. In the acute condition the duodenal mucous membrane around the bile-papilla, and that in the lower end of the common duct, become swollen and edematous and cause partial obstruction of the biliary outlet; this is followed by jaundice of varying intensity. At times a plug of mucus in the diverticulum of Vater is the cause of obstruction. Eppinger, in a case of catarrhal jaundice which came to autopsy as the result of

an accident eight days after the onset of jaundice, found hyperplasia of the lymphoid tissues of the mucous membrane in the portion of the common duct which transverses the intestinal wall. This had led to complete obstruction of the duct and dilatation of the rest of the biliary system.

Mild jaundice, of the catarrhal type, may be of surgical significance when a complication or sequel of echinococcus cysts, gummata, carcinoma of the liver, etc., where the jaundice results from obstruction caused by pressure on the ducts from without. Mayo Robson (1908) thought that in many cases of catarrhal jaundice the head of the pancreas is inflamed and that the pressure of this upon the common bile-duct is sufficient to cause obstruction. These cases, however, usually are more properly classed as chronic catarrhal cholangeitis.

Acute catarrhal cholangeitis is of interest to the surgeon from a diagnostic standpoint. In the early stages, as a rule, the condition is hidden by the usual symptoms of gastro-intestinal catarrh, such as loss of appetite, coated tongue, foul breath, headache, nausea and vomiting. The temperature may rise to 100° F. or 101° F., or higher in rare cases, the patient also having rigors and sweats. Jaundice appears in from two to seven days, being first noticed in the sclera. With the onset of jaundice, as a rule, the temperature falls to normal or even subnormal; the stools become clay-colored, and the urine scanty and highly colored. The liver may be enlarged, but usually is not tender.

The *diagnosis* should be made from the age of the patient, the history of antecedent gastro-intestinal disturbance, the mildness of the attack, and the usually mild course which the disease pursues. Jaundice in middle life or in the aged is generally due to gall-stone disease, pancreatitis, or malignacy. Pain is more marked in these latter conditions, and usually is remittent in type; the jaundice varies in intensity unless there is obstruction of the common duct; and periodic attacks of chills and fever are much more common than in catarrhal jaundice.

The *prognosis* is good. Most cases of acute catarrhal cholangeitis are prolonged over a period of from three to four weeks, the patient ultimately making a complete and permanent recovery.

The *treatment* is purely medical, being directed to the condition of the gastro-intestinal tract. Disappearance of the jaundice indicates return of the bile-passages to normal.

Chronic catarrhal cholangeitis results from mild infection of the bile-tract, or from repeated attacks of acute infection. It is most commonly associated with cholelithiasis (see page 460) although it may occur as an independent affection.

In chronic catarrhal cholangeitis there is edema and swelling of the mucous membrane with increased secretion of mucus. New fibrous connective tissue may be formed in the walls of the ducts, especially in those that are extrahepatic, as the result of round-cell infiltration. The walls usually are thicker than normal. In cases of partial or complete obliteration of the lumen, the ducts above the obstruction become markedly dilated.

The *symptoms* are essentially those of recurring or relapsing jaundice and resemble those of acute catarrhal jaundice extended over a greater period of time. If the obstruction of the common duct is not complete, and if there is no ascending infection of the bile-passages, the disease runs a mild course with remissions in the intensity of the jaundice. There usually is no enlargement of the liver or spleen.

It is almost impossible to make a correct differential *diagnosis* in all cases. The conditions most frequently simulating chronic cholangeitis are calculous obstruction of the common duct, pancreatitis with obstruction of the bile-passages, or malignancy; and these often are the cause or the remote result of the biliary infection. In cases where the condition appears to be a continuation of a simple catarrhal jaundice, the diagnosis may be made, but with only a fair degree of certainty on account of the probability that the persistence of symptoms is due to a stone in the common duct.

The *prognosis* is modified by the associated lesions.

The *treatment* is the same as that in acute catarrhal jaundice, except in prolonged cases when the bile-passages should be explored and drained. Operation is indicated on account of the impossibility in many cases of excluding gall-stone obstruction. The operation of choice is a cholecystomy.

Suppurative Cholangeitis.—This rarely is dissociated from antecedent lesions of the biliary tract, the most common of which are gall-stones and tumors causing obstruction of the ducts. Any condition which interferes with the normal flow of bile reduces the resistance of the ducts and makes them more susceptible to infection. The active etiological factor is always bacterial life (page 416). The result of the infection generally is widespread, although in a few instances it may be limited to the ducts themselves. As a result of the obstruction below, there is a dilatation of the ducts, which usually are filled with bile-stained purulent material. The mucous membrane is congested and edematous; the walls of the ducts are infiltrated, softened, and much thickened. On the surface of the mucous membrane may be seen

points of ulceration. The liver usually is enlarged and softened, and often is the seat of abscesses which usually are found near the ends of the radicles of the hepatic ducts and vary considerably in number and size (*suppurative hepatitis*, page 450). In some instances the outward pressure of the collections of pus found in the substance of the organ renders the surface of the liver irregular. In advanced cases the abscess or abscesses reach the surface and cause infection of the serous covering with a resulting perihepatitis, or peritonitis. Involvement of the pleura and lungs sometimes follows, with or without the development of a subphrenic abscess.

The onset often is insidious, and the *symptoms* may not even suggest the serious underlying condition. There is generally a history of an antecedent infection either of the biliary passages (cholelithiasis, chronic cholangeitis), or of the general system, such as typhoid fever, pneumonia, etc. In those cases where there has been jaundice, serious infection of the biliary passages is announced by chills, fever, and sweats, which may be very severe. Generally there is loss of appetite, nausea and vomiting, and progressive emaciation. Jaundice is present in the majority of cases, but may be entirely absent where no complicating lesions exist.

Pain of a dull aching character almost always is present. Sharp pain, severe in character, is present in those cases where there is an associated cholecystitis, cholelithiasis, obstruction of neoplasm, etc. The liver, spleen, and gall-bladder usually are enlarged, and there is tenderness of the liver and over the gall-bladder region.

Examination of the blood shows leukocytosis with marked increase in the polynuclear cells. The increase in the leukocytes often varies considerably during the course of the disease, being greater during and after the chills and fever.

Extension of the process causes involvement of the surrounding structures; pleurisy, pericholecystitis, *multiple abscesses of the liver*, etc., are among the usual complications. The symptoms of cholangeitis then are more or less hidden by the more severe symptoms of the complicating condition.

A *diagnosis* of suppurative cholangeitis may be made safely in those cases where chills, fever, sweats, jaundice, enlargement of the liver, gall-bladder and spleen, and a high polynuclear leukocytosis are found, in connection with some antecedent disease of the biliary tract. Suppurative cholangeitis may be differentiated from tropical abscess of the liver by the history of dysentery in the latter affection. In malaria the chills and fever are of more regular periodicity and

blood examination should show the presence of the plasmodium and the absence of a leukocytosis.

The *prognosis* in suppurative cholangeitis is greatly modified by the treatment instituted, unless nature anticipates the surgeon and drains the bile-ducts through a fistulous opening between the gall-bladder or common duct and some viscus of the abdomen or thorax. Without drainage of the bile-ducts the prognosis is very unfavorable.

The *treatment* should be prophylactic by treating carefully and persistently any condition of the gall-bladder or biliary passages that might allow a subsequent virulent infection of the biliary tract. Gall-stones should be removed whenever the fact of their presence has been established (page 495). After the onset of suppurative cholangeitis, the biliary tract should be drained by means of a cholecystostomy or a choledochostomy (Chapter XXIV).

Immediate operation offers the best prognosis in cases of *acute obstruction of the common duct* from suppurative cholangeitis, as it does in the same condition produced by impaction of a calculus. By this means further infection of the liver is prevented, and jaundice is arrested before it becomes so profound as to cause deterioration of the blood.

Cholecystitis.—Stagnation of bile in the gall-bladder is the main predisposing cause of infection. Hence **the stagnant gall-bladder** may assume almost the importance of a clinical entity. Owing to the obstruction to the discharge of the bile from the gall-bladder its muscular tunic hypertrophies, the mucosa becomes somewhat thickened, and there is in the submucous, muscular and subserous coat a moderate lymphocytic infiltration. According to Aschoff and Bacmeister (1909), this infiltration is not an evidence of infection but is due to increased absorption from stasis. The stagnant gall-bladder may or may not contain a calculus; usually a solitary stone is present, of pure cholesterin, formed of radially disposed crystals. The stagnant gall-bladder is filled with thick, viscid, non-labile bile, which is very dark olive green, sometimes nearly black in color.

The lesions found in cholecystitis vary from a very mild catarrhal involvement to the most virulent phlegmonous or gangrenous type. The various degrees of inflammation may be classified as catarrhal, suppurative, phlegmonous, and gangrenous, the result of the infection varying with the virulence rather than with the variety of the invading bacteria. It was suggested by Kelly, however, that "the milder catarrhal lesions are most commonly due to the typhoid bacillus and the colon bacillus, and the suppurative lesions to the pyogenic cocci."

In **acute catarrhal cholecystitis** the mucous membrane is edematous and swollen, especially in the region of the neck of the gall-bladder; and the mucous membrane of the cystic duct is involved to a greater or less degree, with consequent narrowing of the lumen of that channel. The walls of the gall-bladder are thickened and edematous and distention by retained material renders them tense. The thickening is due, according to Aschoff and Bacmeister, chiefly to inflammation of the subserous tissues of the gall-bladder. The contents of the gall-bladder usually consist of bile which is thick and tarry, or of serous fluid which is bile stained. This acute inflammation may subside quickly with drainage through the cystic duct, in which instance all symptoms will disappear very rapidly. In other cases the inflammatory process continues, producing first the suppurative and later the phlegmonous forms of cholecystitis. Sometimes as the acute process subsides, a chronic catarrhal inflammation takes its place, often with the formation of gall-stones; or acute exacerbations may occur which finally may result in gradual obliteration of the cystic duct and hydrops of the gall-bladder.

Hydrops of the gall-bladder (*hydrops vesicæ felleæ*) may be transitory or permanent. As pointed out by Kehr (1901), acute infection of the gall-bladder and cystic duct of a mild character usually is accompanied by hydrops. Upon the subsidence of the inflammation, the cystic duct recovers its function, drainage of the gall-bladder occurs and the hydrops disappears. In these cases there is an edema of the mucous membrane of the cystic duct, the valves of Heister formed by the convolutions of the mucous membrane being so swollen that obstruction results. With increased inflammation there may be superficial ulceration, and as a result of the cicatricial contraction of the walls of the duct, the lumen may be encroached upon to such an extent that there may be complete occlusion, with permanent hydrops.

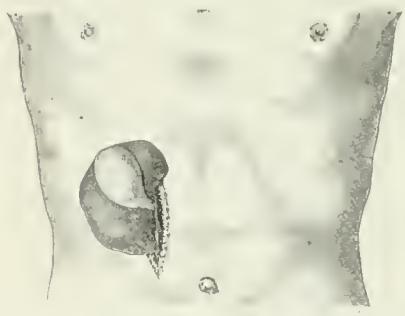


FIG. 137.—Hydrops of the Gall-bladder. From a Patient in the Lankenau (formerly German) Hospital.

The experimental work of Segré (1906) proved that very marked changes occur in the walls of the gall-bladder from obstruction of the cystic duct. There is first a degeneration of the epithelium; this is followed by degeneration of the muscular layer; finally, all

the layers become transformed into connective tissue. When stenosis of the duct is incomplete the muscular coat hypertrophies; in complete occlusion it atrophies and degenerates, so that the gall-bladder walls may become very thin, even translucent, and having the appearance of parchment.

The contents of the gall-bladder usually consist of a clear mucoid fluid, the secretion of the mucous membrane.

The size of the dropsical gall-bladder depends to a great extent upon the result of previous infections of this viscus and the presence or absence of pericholecystic adhesions from a prior inflammation. When the gall-bladder walls have not undergone cicatricial contraction from previous inflammatory attacks and when not bound down by surrounding adhesions, the gall-bladder may distend until it reaches the brim of the pelvis. If adherent there will be less chance of expansion; indeed it must not be forgotten that in addition to the secretion of the mucus, with which a dropsical gall-bladder is filled, there may also be a certain amount of absorption going on in the gall-bladder, and that if the processes of secretion and absorption are nearly equally balanced the gall-bladder may not become distended.

A dropsical gall-bladder is a constant menace to the patient, as it may become infected, resulting in secondary empyema of the gall-bladder; while if of large size it is exposed to external injury.

Suppurative cholecystitis, or empyema of the gall-bladder, may result from a pre-existing *hydrops vesicæ felleæ*; usually, however, it arises as a consequence of a virulent infection in a gall-bladder which previously had been fairly healthy, forming a more advanced stage of the so-called acute catarrhal cholecystitis already described. The condition is comparable to a suppurative appendicitis; the mucous membrane is much congested and edematous, and may be ulcerated; there are minute miliary abscesses in the submucous, muscular and subserous coats; the cystic duct becomes quickly occluded; and distention of the gall-bladder ensues. G. G. Ross operated upon a patient in the Lankenau (formerly German) Hospital, in whom the distended gall-bladder, full of pus, reached to the brim of the pelvis. The condition had been mistaken for an ovarian cyst. Such extreme degree of distention is very rare. E. M. Foote (1910) collected five cases where the gall-bladder filled almost the entire abdominal cavity (Terrier, Tait, Gersuny, Erdmann, and Collinson).

As the pressure increases the pus seeks an exit for itself through the cystic duct; occasionally it is discharged in this way. Usually, however, unless relieved by operation, the condition of the walls of

the gall-bladder is such that perforation takes place, with or without the previous formation of pericholecystic adhesions, just as in the phlegmonous and gangrenous types of cholecystitis presently to be described. Very rarely the inflammation subsides, the micro-organisms lose their virulence and die, and the empyema is converted into a hydrops with sterile or very slightly infectious contents.

Pericholecystitis most frequently is caused by suppurative cholecystitis.[1] As the purulent infiltration spreads through the walls of the gall-bladder, localized peritonitis occurs, usually as the result of perforation of miliary abscesses in the subserous coat. The gall-bladder becomes adherent to the colon, duodenum, stomach, omentum, or even to the small intestines; and occasionally *suppurative* pericholecystitis is found without any macroscopic perforation of the gall-bladder. These pericholecystic adhesions serve as a protection against the development of diffuse peritonitis; but as the acute inflammation subsides the adhesions contract, cause distortion of the gall-bladder and bile-ducts, obstruction of the pylorus or colon, and thus predispose to recurrent attacks of cholecystitis, to intestinal obstructions, etc.

Biliary Peritonitis Without Perforation of the Bile-passages.—This is a condition occasionally seen, and requiring a few words of explanation. A bile-stained peritoneal effusion is found, and yet no macroscopic evidence of a perforation can be discovered anywhere in the bile-tract. G. G. Davis called attention to the condition in 1907, reporting two cases of bile-stained peritoneal effusion without apparent lesions of the biliary tract. In one of these cases chemical examination failed to give any evidence of the presence of bile in the bile-colored peritoneal exudate, though the gauze sponges were stained characteristically yellow. Clairmont and Haberer reported a case with similar effusion, but with a stone in the common duct, yet no perforation; and Schievelbein has reported another case, in which operation was done by Ritter. In none of these cases, however, was the fluid examined for bile. Wolff has reported three more cases, and the whole subject has been reviewed by Johansson (1912) and Buchanan (1917).

Clairmont and Haberer (1910) produced the condition experimenally in a few dogs out of a large number in which they caused obstruction of the common duct for another purpose: in a few days these dogs had biliary peritonitis; the abdominal wall was markedly icteric, while the skin and the rest of the muscular tissues showed only slight jaundice.

[1] It is desirable to distinguish between *pericholecystitis*, an active inflammatory state, and *pericholecystic adhesions* the result of previous inflammation.

Schievelbein (1910) claimed that this biliary effusion is a filtrate through the walls of a nearly normal gall-bladder. In a gall-bladder altered by disease the walls become impermeable for such filtration. He thinks the subserous position of the canals known by Luschka's name ("Luschka's Gänge") has much influence in permitting this filtration. According to Johansson, these canals exist only in about 10 per cent. of gall-bladders. It thus appears that only when the two rare factors coexist (presence of Luschka's Gänge, and acute obstruction of an almost normal gall-bladder) can biliary peritonitis occur without perforation of the bile-tract. That the effusion in some cases at least really is biliary is demonstrated by the cases of Schievelbein, and the similar case mentioned by Gibbon (1913), in both of which the bile-stained fluid oozed through the walls of the gall-bladder under the operator's eyes.

Buchanan, in his admirable study of the subject (1917), concludes that with our present knowledge it is not possible to classify all cases of this nature in the same category; many of them he believes are due to retroperitoneal ruptures or perforations of the bile passages, the effused bile leaking into the peritoneum some distance away from the actual perforation which thus is readily overlooked.

The *treatment* consists in evacuating the effused bile by mopping, after laparotomy, and in drainage of the pelvis; where not contra-indicated, the common bile duct itself should also be drained as advised by Buchanan.

In **phlegmonous chlecystitis** there is a still more virulent infection. The mucous membrane becomes markedly edematous and swollen, the submucous, muscular, and subserous coats become riddled with miliary abscesses, resulting in separation of the various coats from each other and sometimes in the separation of the entire mucous membrane as a slough. The walls of the gall-bladder are dark in color, rigid, but friable; the contents are bloody pus, and sloughs. Usually the process is so acute that no pericholecystic adhesions are formed. The necrosis extends through the serous coat, a layer of lymph alone separating the slough from the peritoneum; when this gives way (**perforation**) the highly infectious contents are poured out into the unprotected peritoneal cavity, and diffuse peritonitis ensues.

Gangrenous cholecystitis results from the most virulent infections, or from thrombosis of the nutrient artery. The lesions are practically the same as those of the phlegmonous type except that the necrosis or gangrene involves a considerable portion of the viscus, instead of being localized to the area of perforation. Owing to the poorer blood

supply at the fundus, the gangrenous process usually starts there and extends toward the cystic duct. Many so-called cases of gangrenous cholecystitis, really should be termed ulcerative cholecystitis, a condition which usually forms a part of suppurative or phlegmonous inflammation, as already described.

Volvulus of the gall-bladder is rare. The presence of a mesentery to the gall-bladder, resulting in the condition known as *wandering gall-bladder* (page 444), is a predisposing cause. The existence of a volvulus seldom is recognized during life; if any localizing symptoms are present they usually are misunderstood. Kübig (1912) records one case found at autopsy, and refers to three other cases reported by Müsham, Mayer, and Fischer, in which the diagnoses made had been appendicitis, hydronephrosis, and gall-stones. All these patients were old, and three of them were women. In many cases gangrene of the gall-bladder results from interruption of the circulation. One case of torsion of the gall-bladder has been encountered at autopsy at the Lankenau Hospital:

Postmortem Record: J. McM., male, death from endocarditis. The gall-bladder was distended, its walls thickened and adherent to the liver. The cystic duct was obstructed by torsion. There was no stricture of the duct. No calculi were present.

Non-calculous cholecystitis as a pathological entity is a condition found in about 15 to 20 per cent. of all diseases of the biliary tract. In a series of 476 patients with diseases of the biliary tract operated upon by the senior author, gall-stones were absent in 18.3 per cent. Riedel found no stones in eleven of sixty-five patients operated upon under the diagnosis of cholelithiasis.

The etiological and pathological factors in cholecystitis have been considered at page 418.

The **symptomatology of cholecystitis** is most variable. In many instances the attack is so fleeting that the patient does not remember it and it can be recalled to his mind only after very close questioning. In other instances the onset of the inflammation is sudden and the symptoms alarming. The wide variance between these two conditions is due entirely to the virulence of the invading micro-organism and the presence or absence of an infectious disease such as typhoid fever (page 430).

The *pain* may be dull and aching, severe and continuous with acute exacerbations, or paroxysmal, being then identical with the so-called gall-stone colic. Solieri (1911) has reported the case of a

patient (who had recently had typhoid fever) whose biliary colic was caused by *hemorrhages into the gall-bladder;* and a patient in whom intracystic hemorrhage seems to have been responsible for attacks of biliary colic, was under the care of the junior author at the Episcopal Hospital in 1911. This patient's history was recorded in the first edition of this book.

The initial pain usually is in the epigastrium, or it may be diffuse, gradually becoming localized in the right hypochondrium. The pain may remain localized or may be referred to the right shoulder, the right shoulder-blade, or to the right iliac fossa, in the latter instance simulating the pain of appendicitis with which condition cholecystitis may be associated.

Nausea and vomiting vary greatly. If the onset is violent there will be marked nausea and vomiting accompanied by great prostration. In mild cases there may be slight nausea and no vomiting. When the vomiting persists, the inflammation of the gall-bladder, in most instances, has extended beyond the confines of that viscus with resulting pericholecystitis or peritonitis.

Fever usually is present but varies in degree and persistency. When the infection of the gall-bladder complicates some other disease, such as typhoid fever, the temperature may be attributed to the underlying disease; if it arises during the convalescence from typhoid fever the temperature may be high from the effects of the cholecystitis alone.

Muscular rigidity always is present, in some cases being of such a character that it will simulate a tumor. Careful palpation sometimes reveals the true gall-bladder tumor lying beneath the rigid muscle.

Tenderness usually is marked, and especially so in the presence of pericholecystitis. The tenderness in the beginning of the affection may be more or less diffuse, although it will almost invariably become localized over the gall-bladder.

The *gall-bladder will become enlarged*, the size of the viscus varying greatly in different cases. In some instances it is so large that it is distinctly visible. It is uniformly pear-shaped, and moves with respiration. It may be displaced by manipulation in those cases where there are no adhesions, but always returns to its former position, thus differing from a movable kidney or a pyloric tumor. As the gall-bladder increases in size, it usually extends downward and inward toward the umbilicus; rarely it may be found in the right flank, in the right iliac fossa, or even extending to the brim of the pelvis. The subsequent history of the enlarged gall-bladder will depend upon

the condition of the lumen of the cystic duct and the presence or absence of virulent infection. In the majority of cases, the cystic duct becomes patulous with the subsidence of the inflammation and the gall-bladder drains itself through the natural channels; with obstruction of the cystic duct persisting, either a hydrops or an empyema of the gall-bladder will result.

Jaundice is never seen in uncomplicated cases of cholecystitis. As long as the common and hepatic ducts remain patulous, jaundice does not occur. If the inflammation should involve either of these ducts directly or through adhesions, jaundice would ensue as soon as the lumen of the ducts became greatly lessened.

The *results of acute cholecystitis* are many; most of them are of decided clinical importance as they have a great bearing on the subsequent health and activity of the patient. In a large majority of the cases the symptoms presented disappear in from four to ten days, but it is probable that the gall-bladder seldom, if ever, returns to the condition in which it was before the infection. A chronic inflammation may ensue (page 459). A very frequent consequence of a mild, subsiding infection of the gall-bladder is the formation of gall-stones (page 420). Sometimes the symptoms that persist after the subsidence of the inflammation are due not only to the condition of the gall-bladder, but to adhesions formed between that viscus and adjacent organs, the result of a pericholecystitis (page 452).

When the infection is of a very severe type, or where it does not subside but rather becomes augmented, possibly by added infection, much more serious conditions of the gall-bladder ensue, *suppurative*, *phlegmonous*, or *gangrenous cholecystitis*. Of these varieties of gall-bladder disease the gangrenous is the most rare, while the suppurative type is not uncommon. In most instances the severe forms of infection occur in connection with cholelithiasis. In a series of 328 operations on the biliary tract by the senior author, gangrene of the gall-bladder was encountered only twice. Both patients recovered.

The symptoms presented by these severe forms of cholecystitis are similar to those of acute cholecystitis, but greatly aggravated, with much more pronounced indications of involvement of the peritoneum. The pain and tenderness are more general, at times extending over the entire abdomen. Nausea and vomiting may be persistent, and the picture presented is one of infection of the general peritoneal cavity rather than of the gall-bladder. The temperature is higher, reaching 103° or 104°F. The blood count shows a decided increase of leukocytes with relative increase in the polynuclear cells.

Perforation of the gall-bladder may cause a fistula between the gall-bladder and the stomach, duodenum or colon; it may open into the liver with the formation of a liver abscess; it may perforate into a mass of adhesions surrounding the gall-bladder with the formation of a local abscess. In such cases the acute symptoms are suddenly relieved. But in many cases perforation occurs into the general peritoneal cavity, and cause rapidly spreading and usually fatal peritonitis.

The **diagnosis** of cholecystitis usually is readily made. The chain of symptoms presented in most cases, (nausea and vomiting, pain and tenderness over the gall-bladder region, rigid abdominal muscles over an enlarged and tender gall-bladder) should lead to a correct diagnosis. The mild and severe forms of cholecystitis generally may be differentiated by the intensity of the symptoms presented. It is generally impossible to differentiate between calculous and non-calculous cholecystitis, especially when the gall-stones are confined to the gall-bladder. Gall-stones, as a rule, give rise to no symptoms when in the gall-bladder, the phenomena of gall-stone colic being due to inflammation consequent upon infection. Gangrene or perforation in acute cholecystitis cannot always be recognized but may be suspected on the occurrence of a sudden exacerbation of the pain and increase of the general peritoneal symptoms; and perforation especially may be suspected if a gall-bladder previously palpable suddenly seems to disappear.

Acute cholecystitis must be differentiated from acute *appendicitis* although failure to do this is not uncommon. The pain of appendicitis may be in the gall-bladder region, and the pain of the cholecystitis may be found in the right iliac fossa. Tenderness from an inflamed gall-bladder may be elicited in the appendiceal region. Both diseases may be caused by a common bacterial invasion. In patients past forty-five years of age appendicitis is comparatively rare. The pain of acute cholecystitis is more often limited to the epigastrium and the right hypochondrium, and that of acute appendicitis more often to the right iliac fossa. Referred pain is not common in appendicitis, while it often is present in cholecystitis. The initial pain in appendicitis is more general than that of cholecystitis. The presence of a tumor which moves with respiration is characteristic of cholecystitis. We agree with Hotchkiss (1899) in attaching great importance to lateral compression of the lower ribs in developing pain in cholecystitis. A careful study on the onset will be a great help in clearing up the diagnosis.

In *intestinal obstruction* there is a chain of symptoms similar to those of acute cholecystitis, pain, nausea and vomiting, constipation and tympany. But in intestinal obstruction there is no rise of

temperature before the onset of peritonitis; while in acute cholecystitis elevation of the temperature is common. If a gall-bladder tumor can be recognized the diagnosis is clear.

Acute pancreatitis may give rise to the same symptoms as a severe infection of the gall-bladder. In pancreatitis, however, there generally is much more constitutional disturbance and the patient appears to be more profoundly ill than in acute cholecystitis. The pain and the tenderness in pancreatitis more often are in the epigastrium. Tumor is more commonly recognized in gall-bladder infection and is located in the majority of the cases beneath the ninth costal margin, while that of the pancreas usually is behind the stomach, near the mid-line or to the left, and usually not easily palpable. Some writers claim that differentiation between these two affections is of academic interest only, as the treatment they advocate for both conditions is the same, immediate drainage of the gall-bladder. We do not agree with this opinion in all cases, however, as better results often may be obtained by proper treatment before operative measures are instituted.

Prognosis.—The prognosis in the majority of cases of acute cholecystitis is good, the inflammation subsiding and the gall-bladder draining itself through the cystic duct. But a gall-bladder that has been infected will be the seat of repeated attacks of inflammation in a majority of cases. In the severe types of infection the prognosis must always be guarded, and in the gangrenous or phlegmonous type it is very grave. The sequels of the inflammation often are of much more importance than the original attack, pericholecystic adhesions frequently causing great impairment of health with marked suffering; while gall-stone formation is an exceedingly frequent sequel of infection of the biliary tract. The ultimate prognosis in these cases is greatly modified by the treatment instituted; this we believe should be operative interference, the surgical procedure being modified by the conditions found.

Treatment.—The majority of mild infections of the gall-bladder will subside under appropriate medical treatment. This implies rest in bed in the semi-sitting position, hot or cold applications, preferably the latter, to the upper right quadrant of the abdomen, absolutely nothing by mouth and fluids by proctoclysis. Before starting proctoclysis give a cleansing enema; this can be repeated each day if the patient suffers with accumulation of gas. To the enema may be added asafetida or glycerine and turpentine. When all acute symptoms have been absent for thirty-six or forty-eight hours sodium phosphate in hot water may be given by mouth. The treatment recently advo-

cated by Lyon (1920), consisting in the local application of magnesium sulphate to the duodenal mucosa by means of the duodenal tube, so as to cause relaxation of the sphincter of Oddi, can, so far as we are able to see, have no influence on the gall-bladder and cystic duct when they are acutely inflamed.

The treatment outlined above is not that usually instituted by internists, but it is our opinion that this is the rational course to pursue, with various modifications, in the early stages of acute cholecystitis. We know that patients recover more rapidly and with less discomfort, *if all food is withheld for from forty-eight to seventy-two hours*, as above stated, or until there is restoration of peristalsis and the passage of gas. Repeated lavage of the stomach relieves that organ of any material regurgitated into it, and checks the nausea and vomiting. We prefer ice-cold applications to the gall-bladder region, as these allay pain and inflammation more readily than applications of heat. After recovery from the attack, treatment at some of the noted Springs, such as Richfield, Mt. Clemens or Saratoga, may be of value, if the case is one of non-calculous cholcystitis.

Operative procedures should be instituted in all recurrent cases of mild inflammation of the gall-bladder. A second attack of non-calculous cholecystitis is an indication that the gall-bladder is the seat of a chronic infection which is prone to flare up at any time and result in much more severe consequences. Acute cholecystitis of a severe type should be operated upon as soon as possible, except in the presence of diffuse peritonitis, when better results will be obtained by the so-called Ochsner treatment for peritonitis.

The operation of choice in non-calculous cholecystitis is simple drainage of the gall-bladder. If there is obstruction of the cystic duct due to inflammatory thickening of the mucous membrane, drainage of the gall-bladder will allow the inflammation to subside and will thus restore free communication between the gall-bladder and the common duct.

In the more severe types of non-calculous inflammation, where there is evidence that the gall-bladder will become functionless, or where the cystic duct is permanently closed by cicatricial contraction, or where there is decided enlargement of the lymph-node at the junction of the supra- and retroduodenal portions of the common duct, indicating infection of the chain of peri-pancreatic lymphatics, cholecystectomy should be performed.

Chronic cholecystitis may be the end result of acute inflammation, or it may be due to an inflammation of the chronic type from the

beginning. Non-calcuous chronic cholecystitis is not so common as the calculous form of the disease, gall-stones being associated and sustaining the inflammation in a large majority of cases. The etiology of this condition is that of the acute inflammation, "the chronicity," as stated by Kelly, "being a manifestation of lingering infection (which is common), or the consequence of very low-grade infection with almost but not quite sufficient biliary drainage."

In non-calculous chronic cholecystitis, the lesions may be confined entirely to the mucosa, with congestion, swelling, desquamation and a greater amount of mucus than normal; or there may be infiltration of the walls of the gall-bladder, with areas of erosion. When the tips of the villi which have undergone desquamation become stained with bile, what has been called the "strawberry gall-bladder" is present (p. 466); the same condition may be due to deposition of lipoids and cholesterin in the mucosa and submucosa (Reiman, 1919). In other cases the walls of the gall-bladder are distended and thin and the gall-bladder is filled with thick mucus and bile. In long-standing cases there usually is contraction of the organ; associated with these cases very frequently is a mass of adhesions surrounding and pressing upon the gall-bladder.

Chronic cholecystitis is almost always present in patients with biliary calculi which give rise to symptoms. We believe that the symptoms are caused by the inflammation and not by the gall-stones themselves. When gall-stones are present the changes in the walls of the gall-bladder usually are more marked, the inflammation invading the deeper layers of the walls and causing hyperplastic and proliferative changes. This condition is considered by Kelly as the beginning of carcinomatous degeneration.

The **symptoms** presented by chronic cholecystitis, whether calculous or non-calculous, are essentially those of cholelithiasis (page 481), and it is impossible to determine from symptoms alone whether or not calculi are present. Repeated attacks of biliary colic followed by icterus, might lead to a correct diagnosis of cholelithiasis. In the patients of the senior author at the Lankenau Hospital, colicky pains have been noted in 70.6 per cent. of the cases of non-calculous chronic cholecystitis, while jaundice has been present in 35.3 per cent.

The *prognosis* and *treatment* are those of cholelithiasis (page 495).

Cholelithiasis

The etiology of gall-stone formation, the character and composition of gall-stones, etc., have been considered in the previous chapter (page 419).

Pathology.—In studying the pathology of cholelithiasis, it probably would be more correct to classify the various lesions as complications of gall-stones, rather than as changes due to the mere presence of the calculi. Gall-stones, in the absence of an added infection, seldom cause any serious pathological changes; they may remain indefinitely in the gall-bladder, where they have been formed, without giving rise to any symptoms directly referable to the biliary tract. Kehr (1901) stated that almost every tenth adult has gall-stones but that only 5 per cent. of those having biliary calculi present signs or symptoms of their presence, the remaining 95 per cent. having no knowledge of their existence. The postmortem records of the Lankenau Hospital show the presence of calculi in the gall-bladder or ducts in over 11 per cent. of the autopsies, the death in each instance having been due to diseases other than those of the biliary tract. Although these gall-bladder examinations made at autopsy were limited to 524 cases, yet the findings support the statement of Kehr so far as the prevalence of biliary calculi is concerned. The proportion of those having gall-stones and not presenting any symptoms may be as high as Kehr states; and the presence of calculi in the gall-bladder may be of no significance; yet we believe that a more careful study of such patients would elicit symptoms, in the majority of cases, due to the presence of gall-stones but incorrectly assigned to functional disturbances of the gastro-intestinal tract.

When the gall-stones are formed there are present in the gall-bladder bacteria of attenuated virulence, and a catarrhal inflammation of the mucous membrane. The gall-bladder rids itself of the catarrhal inflammation and bacteria, and the normal flow of bile becomes reestablished in a large proportion of cases, leaving the gall-stones behind as a record of the acute process. In such instances the stones may remain in the gall-bladder indefinitely; and by themselves, unaided, will not necessarily cause any further serious trouble. The persisting lesions are so slight that they give rise to no symptoms or signs in the gall-bladder region; but there are numerous instances where disorders of digestion can be cured only by the removal of gall-stones, the presence of which has never been suspected by the patient. We believe that a recognition of these cases will greatly decrease the estimated proportion of those individuals having biliary calculi but not presenting symptoms of their presence.

Calculi in any portion of the biliary tract invite renewed infection. Without such infection they seldom give rise to serious pathological lesions, and the gall-bladder and ducts may remain for long periods in

the condition they were in after the subsidence of the stone-forming bacterial invasion. Riedel, as quoted by Kehr, was of the opinion that a "foreign-body inflammation," independent of bacterial invasion, may arise in the gall-bladder or ducts with the same effect that would result from a "bacterial inflammation." The "foreign body inflammation," possibly, may occur; it is more probable, however, that in all such instances a bacterial invasion takes place afresh, being so rapid in its course, at times, that it does not give rise to any marked symptoms or pathological lesions.

Non-inflammatory movements of the concretions within the gall-bladder may be caused, to a very slight degree, by the to-and-fro motion of the bile. The lesions thus produced are of little significance, although Kehr claimed that "cancer of the gall-bladder may develop from the irritation of the stones actually lying quiescent in the fundus." Stones may be come dislodged from their resting place and cause some interference with the outflow of the bile from the gall-bladder. As a rule, however, they do not wander; they remain where they were formed, until dislodged by contractions of the gall-bladder incited by inflammatory irritation, the latter being the result of the new infection of the viscus or of an acute exacerbation of an old process.

The mechanical action of the gall-stones after the onset of active inflammation in the biliary tract is that of a foreign body. The concretions may remain quiescent; they may be forced into the cystic duct, or through it into the common duct, and through the common duct into the intestine. After reaching the duodenum they may be expelled with the feces or may become so augmented by added laminations that intestinal obstruction will result. Occasionally gall-stones have lodged in the appendix vermiformis, as in cases reported by Lediard, Budd, Serey, Rehn, Treves, and Robson; and as in a patient under the care of H. C. Deaver. Chemical study of the concretions is requisite to ascertain their origin.

Gall-stones may cause necrosis of the gall-bladder or ducts with the subsequent perforation and expulsion of the stone into the liver substance, into a mass of adhesions formed around the gall-bladder, or into one of the adjacent hollow viscera, such as the stomach, the duodenum or the colon. If they become lodged in the cystic duct, there will result a closure of that channel either complete or partial, with simple hydrops or empyema of the gall-bladder, depending upon the nature and virulence of the invading organism. At times the inflammation will subside and the cystic duct remain partially patent, so that there will be a discharge of the contents of the gall-bladder and a return to com-

paratively normal conditions. The presence of the stone in the duct will encourage subsequent infections, as it will always act as an irritant; it is less apt to do this if it becomes encysted by being covered by the swollen mucous membrane or by forming a pocket or diverticulum for itself. The effect of such improvement of the stone is to cause a deviation of the channel and consequent interference with the natural to-and-fro motion of the bile.

The stone may become lodged in the common duct and may remain there an indefinite period without giving rise to any symptoms. On the other hand, it may cause complete and temporary or partial and permanent obstruction of the duct, with damming up of the bile, changes in the gall-bladder and liver, intermittent icterus, etc.

In a series of 549 cases of cholelithiasis, operated upon by the senior author at the Lankenau (formerly German) Hospital, the concretions were found in the following locations:

In the gall-bladder alone in	316 or 57.5	per cent.
In the gall-bladder and cystic duct	63 or 11.4	per cent.
In the gall-bladder and common duct	60 or 10.9	per cent.
In the gall-bladder and hepatic duct	1 or 0.18	per cent.
In the gall-bladder, cystic duct and common duct	3 or 0.5	per cent.
In the gall-bladder, common duct and hepatic duct	4 or 0.7	per cent.
In the cystic duct alone	41 or 7.4	per cent.
In the cystic and common duct	1 or 0.18	per cent.
In the common duct alone	37 or 6.7	per cent.
In the gall-bladder, cystic duct, common duct, and hepatic duct	5 or 0.9	per cent.
In the common and hepatic ducts	10 or 1.8	per cent.
In adhesions surrounding the gall-bladder	4 or 0.7	per cent.
Location of stone not mentioned in	4 or 0.7	per cent.

Of far greater importance than the mere presence of the stones in any case of cholelithiasis is the infection that may be added. While it is probably true that a number of those patients who possess gall-stones never present symptoms severe enough to call their attention to the biliary tract, it is undoubtedly a fact that these concretions render persons so affected more liable to infection of the biliary tract than are those who are free from gall-stones. Of 476 patients with disease of the biliary tract, operated upon by the senior author, 389 or 81.7 per cent. had gall-stones. When infection is added to existing biliary calculi, the resulting pathological lesions will depend upon the virulence of the invading micro-organism and the modifying resistance of the patient. They will vary from a mild condition of catarrhal inflammation to a most rapid, wide-spreading gangrenous process. Lesions in all parts of the biliary tract and adjacent struc-

tures may be produced which will result in impairment of health if not in the death of the patient. As Kehr says: "Gall-stone disease enters upon a period of latency and can in this quiescence repose until death puts the man to sleep in the eternal rest of the grave."

Any of those lesions discussed in the previous section (Cholecystitis and Cholangeitis) may arise in cases of cholelithiasis; but the pathological processes in cholelithiasis are more or less modified by the previous inflammation of the bile-passages, and by the mechanical action of the calculi. The gross pathological lesions found in a series of 538 cases of cholelithiasis operated upon by the senior author at the Lankenau (formerly German) Hospital were as follows:

Lesions in 538 Cases of Cholelithiasis

	Cases	Per cent.
Cholecystitis, mild or absent[1]	328	60.9
Cholecystitis, acute, with catarrh, ulceration or gangrene[2]	166	30.8
Hydrops of the gall-bladder	26	4.8
Carcinoma of the gall-bladder	7	1.3
Pericholecystitis with abscess	7	1.3
Contracted gall-bladder, embedded in the liver	1	0.18
Cholecysto-gastric fistula	2	0.36
Adenomatous degeneration involving liver	1	0.18
	538	99.82

In a further series of 804 cases of cholelithiasis operated on by the senior author (1912–1920), the lesions were classed as follows:

Lesions in 804 Cases of Cholelithiasis

	Cases	Per cent.
Without acute infection	628	78.1
With: acute cholecystitis	37	4.6
empyema	23	2.8
hydrops	2	0.2
gangrene or perforation	7	0.8
pericholecystic abscess	1	0.1
acute pancreatitis	8	0.9
acute cholangitis	3	0.3
chronic pancreatitis	46	5.7
pancreatic lymphangitis	40	5.0
ulcer, duodenal	7	0.8
ulcer, gastric	2	0.2
	804	99.5

[1] In forty-seven of these cases there was chronic inflammation and contraction of the gall-bladder.

[2] In sixty-four of these cases pus was present in the gall-bladder (empyema).

In the vast majority of cases the attention of the patient is first called to the presence of gall-stones by a new invasion of the biliary tract by bacteria, or by an acute exacerbation of a chronic inflammation. The acute attack may be very mild and fleeting in its course and may subside so rapidly that it may soon pass entirely from the patient's memory. With each attack, however, changes occur in the gall-bladder causing that viscus to pass through the various stages of inflammation until it finally becomes contracted, with marked thickening of its walls. During the progress of these inflammatory processes, the gall-bladder may change very decidedly in shape and size. Distinct pockets or diverticula are formed in some instances or a contracting cicatrix will cause an hour-glass formation; at other times the viscus will almost disappear, its remnant forming a tight capsule or covering for one or more calculi. These anomalies have been discussed at page 443.

In so-called "*simple cholelithiasis*," the symptoms of which are in reality due to a chronic catarrhal cholecystitis, the changes in the walls of the gall-bladder may or may not be very marked. There may be slight enlargement of the organ with some thickening of its walls. The mucous membrane usually is somewhat thickened, possibly edematous and injected. As a rule the bile is thicker than normal, ropy, and dark in color. These conditions are not serious and usually subside so soon as the excitants of the infection, the gall-stones, have been removed. In the absence of operative interference the gall-bladder and cystic duct only in the rarest instances clear themselves of calculi; in such circumstances the bladder and duct may resume their normal condition. Even when, after the subsidence of an exacerbation, the concretions remain in the gall-bladder and cystic duct, or in the gall-bladder alone, the pathological lesions present may be such that they will not for many years give rise again to symptoms pointing to gall-bladder trouble.

The Cholesterin Gall-bladder.—Moynihan has given this name to certain gall-bladders whose walls seem normal on casual inspection, and the mucous lining of which feels healthy to the palpating finger. The contents are the usual dark and tarry bile of the "stagnant gall-bladder;" but on close inspection there are found imbedded in the mucosa quantities of cholesterin, as fine as sand; this infiltration of the mucosa stops abruptly at the cystic duct. Sometimes minute crystals of cholesterin may be seen glistening on the gauze with which the gall-bladder contents have come in contact; and these will give a clue to the true condition of the gall-bladder walls. It will be recalled that accord-

ing to Aschoff and Bacmeister pure cholesterin stones may be formed without bacterial action; and Moynihan considers such gall-bladders as these typical examples of such manufactories. The cholesterin sand cannot be removed. The gall-bladder must be extirpated.

The Strawberry Gall-bladder.—Under this name MacCarty (1910) described a form of chronic catarrhal cholecystitis in which the gall-bladder resembles at first sight the cholesterin gall-bladder of Moynihan. Study, however, shows that the yellow spots seen against the reddish background are not cholesterin deposits, but the tips of villi which have undergone desquamation and then become stained by the bile. According to studies made at the Lankenau Hospital by Reimann (1919), "strawberry gall-bladder" usually is due to deposit of lipoids and cholesterin in the mucosa and submucosa, these materials coming from the disintegration of cells. Very often he found small crystalline masses of cholesterol or lipoids surrounded by foreign body giant cells. We attach no special significance to such changes. These gall-bladders may or may not contain stones; but in either case their removal is requisite to prevent recurrence of symptoms.

Pericholecystitis.—When the infection is of a more virulent type, the inflammation will extend through the walls of the gall-bladder and affect the visceral peritoneum, a *pericholecystitis* resulting. After reaching the peritoneum, the inflammation may extend to all of the adjacent viscera, resulting in adhesions matting together the gall-bladder, the omentum, the colon, the duodenum, the liver, the stomach, and the ileum into an unrecognizable mass. The acute inflammation may again subside but the changes wrought will be lasting. Permanent alterations in the gall-bladder will be noted, and even if the concretions within the gall-bladder remain more or less quiescent after the subsidence of the inflammation, symptoms of distressing nature will persist, due to the adhesions and the effect of their contraction (Fig. 138).

Perforation of the gall-bladder is a rare event in cases of cholelithiasis, probably because the walls of the gall-bladder have become resistant from the long-standing infection, which from the first has been of a low grade of virulence. If, however, a virulent infection is added to a gall-bladder already containing calculi, perforation may occur precisely as in non-calculous phlegmonous cholecystitis (page 453). In the case of calculous cholecystitis, however, the long duration of a mild infection or the recurrence of attacks of subacute inflammation usually has been sufficient to produce marked thickening of the gall-bladder walls (Plate V), as well as pericholecystic adhesions, so that perforation into the unprotected peritoneal cavity is extremely rare.

Gall-bladder Removed for Acute Calculous Cholecystitis, Showing Numerous Stones Ulcerating Through Walls of Gall-bladder. Count Showed a Total of 914 Calculi. Female, 70 Years, Recovery. Path. No. 8022. *Lankenau Hospital.*

Face p. 466

Thus when a perforation occurs, the contents of the gall-bladder, and not infrequently the calculi themselves, are discharged into the surrounding adhesions, forming a **pericholecystic abscess,** or even into some adjoining hollow viscus (stomach, duodenum, colon) if the portion of the gall-bladder affected by perforation has been closely adherent to such organ, and the sloughing or ulcerative process has not been arrested

FIG. 138.—Gall-bladder Distorted by Pericholecystic Adhesions. (*Lankenau Hospital.*)

spontaneously before penetrating the walls of the adherent viscus. Sometimes the gall-bladder ulcerates into the liver, and an intrahepatic abscess results. In a few instances the adhesions will be very slight, a mere "cob-web," and the stone or stones will appear to hang upon the external surface of the gall-bladder (Plate VI). If the perforation has taken place into a mass of adhesions, a swelling will be formed involving any part or all of the upper right quadrant of the abdominal cavity; the inflammation may extend upward toward the diaphragm, forming a

subphrenic abscess;[1] it may burrow toward the right kidney space and simulate very closely a perinephric abscess of renal or spinal origin. Two such cases were reported by Estes (1905): the true origin of the condition was recognized only after exploratory incision. A rather unusual course for the pus to take in these instances was reported by Jones (1907): in this case the pus burrowed through a mass of adhesions into the right kidney space, forming a perinephric abscess; and this abscess opened into the pelvis of the kidney, biliary calculi being passed from the urinary bladder. Communications of the biliary passages with the pleural cavity and bronchi are considered in connection with the subject of biliary fistulæ (page 525).

A large majority of ulcerative perforations of the gall-bladder occur in those cases which have been neglected after the onset of the acute attack. When the infection is very virulent it may be possible for the succeeding steps of inflammation, ulceration, and perforation to occur so rapidly that operative treatment cannot be instituted; but usually the course of the inflammation is slow enough to allow the removal of the calculus or calculi with drainage of the gall-bladder or cholecystectomy. Early operative treatment of gall-stone cases would prevent in the majority of instances the occurrence of perforation of the gall-bladder and its numerous sequels.

CALCULI IN COMMON DUCT; PERFORATION OF GALL-BLADDER INTO LIVER; SUBPHRENIC ABSCESS; ABSCESS IN GASTRO-HEPATIC OMENTUM. DRAINAGE OF GALL-BLADDER AND COMMON DUCT. DEATH FROM SEPTICEMIA

D. E., male, aged forty-one years; admitted to the German Hospital November 9, 1905. Has had stomach and liver trouble at irregular intervals for past twenty years. Attack of heart trouble four years ago. Complains of dull pain below ensiform, which began eleven days ago. Pain referred to back and to right iliac fossa. Vomited once.

Examination.—Liver extends from fourth rib to costal margin. Tenderness in epigastrium and over gall-bladder. Rigidity over gall-bladder. Respirations upper thoracic and principally left sided.

Operation, by Dr. Deaver. Ether anesthesia. Incision splitting fibres of upper right rectus. Adhesions throughout right hypochondrium. Gall-bladder enlarged but collapsed. Communication between gall-bladder and abscess under diaphragm. Abscesses in liver, and pus in gastro-hepatic omentum. Three calculi in common duct. Pus evacuated; calculi removed from common duct by incision; gall-bladder and common duct drained by separate tubes; gauze drain to subhepatic space. Death twenty-five days after operation from septicemia and exhaustion. No autopsy.

[1] This is much more often secondary to suppurative hepatitis (page 540), though Violett, according to Langenbuch (1897), recorded a case without direct invasion of the liver.

Perforation of the gall-bladder *into the free peritoneal cavity* usually results in a rapidly spreading peritonitis. Very seldom does it happen that the extravasated bile is sterile, or so slightly infectious as to produce no acute inflammatory reaction. Garre, in 1904, found reports of about fifty cases of perforation of the gall-bladder or ducts in cases of cholelithiasis: in thirty-three cases the perforation occurred in the gall-bladder, in one in the hepatic duct, in five in the common duct, and in four in the cystic duct; while in the remaining cases the site of perforation was not mentioned. It is our impression that the perforation usually is at or near the neck of the gall-bladder in calculous cases, while in non-calculous cases the perforation is more apt to be near the fundus of the gall-bladder.

Hydrops of the Gall-bladder, already discussed at page 450, also occurs when a calculus becomes so fixed in the **cystic duct** that the lumen of the channel is occluded. The effect on the gall-bladder is the same as when this duct is occluded by cicatricial contraction, but there is much greater likelihood of patency being restored in the former than in the latter case. In some instances the stone will cause sacculation of the duct, the channel being very tortuous. When the obstruction is complete the gall-bladder becomes distended with a clear mucoid fluid, the secretion of the mucous membrane. In cases of hydrops there is seldom more than one calculus present—that causing the obstruction. This is a radial cholesterin stone; for other (secondary) calculi to form it would be necessary, according to Aschoff and Bacmeister, for bile to be present. Since both in experimental ligation of the cystic duct, and in malignant obstruction of the cystic duct (lesions presenting the same mechanical conditions as impaction of a stone) the gall-bladder rarely becomes distended (hydrops), but usually shrivels up, it is clear that in cases where hydrops does occur there must be some factor (in addition to the mechanical closure of the duct) rendering absorption less than secretion—perhaps a low-grade hematogenous infection.

Calculi in the common duct causes pathological lesions that are far more serious than those produced by stones in any other situation. The condition becomes systemic rather than local, from the direct influence on the physiological activities of the hepatic ducts and their radicles, from absorption by the lymphatics along the duct and from the effect on the gastro-intestinal tract by interference with the functions of the pancreas and the intestines. In exceptional cases no systemic effect is produced, the stone remaining in the duct or passing through it into the duodenum without giving rise to other than transitory symptoms, and no serious pathological lesions result.

The relative frequency of stones in the common duct is shown by the table at page 463. Although they were found in the common duct alone in not quite 7 per cent. of cases, yet in over 21 per cent. of all cases there were some calculi in this situation. Robson found calculi present in the common duct in over 39 per cent. of patients under his care. The more careful the search, and the more experienced the surgeon, the oftener will stones be found in the deeper ducts. As Terrier used to say, biliary surgery tends to become more and more "canaliculaire."

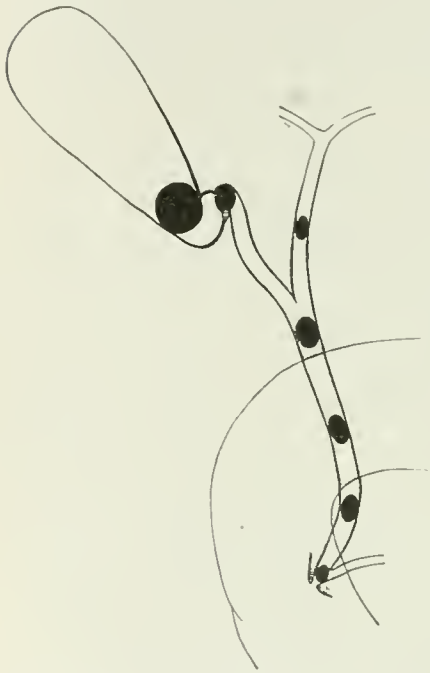


Fig. 139.—Diagram to Show Various Sites of Biliary Calculi.

The position of the stones in the common duct varies from its beginning to its termination in the ampulla of Vater. Courvoisier found the stone, in an analysis of 123 cases,

- At the commencement of the duct in seventeen cases.
- In the middle part of the duct in nineteen cases.
- Near the retroperitoneal part of the duodenum in twenty cases.
- At the ampulla of Vater in forty-one cases.
- Scattered along the entire common duct in twenty-six cases.

Langenbuch said that in two-thirds of the cases the stone is in the duodenal portion of the common duct. A calculus not infrequently projects into the duodenum through the opening of the ampulla of Vater. Calculi in this situation usually are small; indeed Moynihan claims that the further down in the duct the calculus is found, the smaller it is apt to be, the larger stones almost invariably being found in the upper portion of the duct.

As already stated, the vast majority of biliary calculi originally form in the gall-bladder, and are thence discharged into the cystic, and eventually into the common bile-duct; and when the first stone has become impacted in the common duct, all others descending from the gall-bladder will be dammed up behind it, until the complete chain may reach beyond the orifice of the cystic duct and fill the hepatic ducts also. It is of course not impossible, when a stone once has lodged in

the common duct, that other calculi not derived from the gall-bladder may be formed subsequently in the ducts on the hepatic side of the obstruction. Such calculi, however, are almost invariably bilirubin calcium stones, originally of very minute size (sand), which have descended from the intrahepatic ducts, and which subsequently increase in size by lamination.

The number of stones in the common duct varies from one to an indefinite number. The greatest number removed from the common duct by the senior author is 258. The history of this patient is of interest also in showing the size to which the common duct may be dilated.

F. X., female, aged thirty-four years. Admitted to the German Hospital, January 16, 1910. *Family history* negative. *Past history* negative. Has had five children; never has had typhoid fever. *Present illness:* for about nine years has been troubled with indigestion, having epigastric pain with a sensation of stoppage of food in this region; much eructation of gas. Since October, 1907, has been having irregular attacks of pain in right hypochondrium, occurring about once in two months. The pain at times is very severe, radiating to the back and spine, and requiring morphine for its relief. Last attack began January 20, 1910, and then for the first time patient became jaundiced. The pain is unaffected by posture or eating. Has seldom been nauseated, seldom vomited. Since onset of jaundice stools have been clay-colored. Patient was under treatment at Carlsbad for three months last year.

Physical Examination.—Well developed and nourished. Complexion markedly jaundiced, of a lemon tint. Liver dullness extends to costal margin. Mass felt under costal margin in mid-clavicular line, small in size and tender. Slight rigidity. No other mass. No abdominal distention. Hemoglobin 82 per cent.; R. B. C. 3,410,000; W. B. C., 8,150. Coagulation-time ten minutes. Urine contains few granular and hyaline casts. Bile test strongly positive. Cammidge reaction positive. Stool: well-formed masses of fatty consistency, white, acid; occult blood negative; soaps, fatty acid crystals, fat, bacteria, small amount of vegetable material, few leukocytes and epithelial cells.

Operation.—Ether anesthesia. Incision through upper right rectus. Adhesions between gall-bladder and omentum. Adhesions freed and ligated. Gall-bladder incised and fifty-five stones removed. Common duct found to be distended to size of small intestine and filled with bile and calculi. Bile removed with hypodermic needle. Duct incised and 258 stones, varying in size from a millet-seed to a large pea, were found. Obstruction found in ampulla and stone size of hickory nut removed in pieces, with the scoop. Gall-bladder, common and hepatic ducts drained.

Patient made a quick recovery. Drainage fistula completely closed and jaundice was almost absent by time of discharge from the hospital, twenty-seven days after operation.

Complete occlusion of the common duct by a stone is comparatively rare; partial obstruction is much more common. In many instances

the gall-stone remains in the duct without causing any definite symptoms of obstruction, its presence in the duct being discovered only at the time of operation undertaken for suspected stones in the gall-bladder. When obstruction takes place there occurs a damming up of bile with dilatation of the common and hepatic ducts and their radicles. Dilatation of the duct will occur gradually to such an extent that the bile may escape around the stone or stones, with consequent decrease of the jaundice. This intermittent type of jaundice was explained by Fenger (1896) by assuming that the calculus acted as a ball-valve in the common duct, floating loose so soon as a sufficient accumulation of bile had occurred behind it, and again becoming impacted as this bile escaped on the temporary relief of obstruction. But this purely mechanical explanation is invalidated by later operative experience which has shown that in many of such cases, if not in all, the stone is so firmly fixed in the duct that by no stretch of the imagination could it have been assumed to have been acting as a ball-valve.

In the patient whose history is noted above, the common duct equalled the small intestine in size; Langenbuch refers to a case recorded by Schuppell in which the diameter of the common duct was 5 cm.; and Moynihan mentions a specimen in the Museum of Guy's Hospital in which the diameter of the common duct measured 6 inches. This dilatation of the common duct may be saccular, but usually is cylindrical.

Saccular cystic dilatation of the common bile-duct is comparatively rare; the condition sometimes is described as a *cyst of the common bile-duct*. But as McConnell (1920), who has collected 36 cases of this rare condition, points out it really is not a cyst but a diverticulum of the duct, which is a congenital deformity, having been found in the fetus. It is much more frequent in the female than the male, the proportion being about 8 to 1. In the twenty-nine cases collected by Lavenson (1909) the age was stated in twenty-two instances; the average was fifteen years and eight months. Two patients were under one year of age and two were between forty and fifty years of age.

The underlying *cause*, is some form of obstruction or obliteration of the duct on the distal side of the enlargement; for even in cases which are undoubtedly due to a congenital malformation, progressive enlargement of the diverticulum occurs. Congenital obliteration of the common duct (page 441) might give rise to such a cystic formation, were life prolonged a sufficient length of time. In nineteen of the cases collected by Lavenson the causes of the obstruction were as follows: gall-stones in three; papilloma in one; myomyxomatous polyp in one; scirrhous pancreatitis in two; catarrhal cholangeitis in one; a simple

stenosis or obliteration of the peripheral end of the lumen in six; a valve-like fold or angular insertion of the duodenal end of the lumen in five. Lavenson states that in addition to the obstruction of the lumen of the duct, a general weakness of the musculature of the duct is present. Schlössmann (1911) collected sixteen cases of what he considered "idiopathic" cystic dilatation of the common duct.

The dilatation of the duct may vary from that of the index finger to a cyst of enormous size. Weiss (1909) reported a case in which the dilated duct contained 800 c.c. of slightly cloudy biliary fluid. It simulated an echinococcus cyst.

The *symptoms* of cystic dilatation of the common duct are those of obstruction to the normal flow of bile. According to Schlössmann the symptoms presented are merely intermittent jaundice which may continue for years; either colic-like or continuous abdominal pain which seems to vary with the jaundice; and a tumor in the right upper quadrant.

It is practically impossible to make a differential *diagnosis* in all cases. An enlarged gall-bladder or a cyst of the pancreas will present the same tumor-like mass. We strongly advise against exploratory puncture in a suspected case, as adopted by Schlössmann, on account of the great danger of causing peritonitis. Exploratory laparotomy is a much safer procedure.

The *treatment* should be by operation; but the statistics collected and analyzed by McConnell (1920) show a very high mortality. Of thirty-one cases in which operative interference had been instituted, there were 25 deaths; none of the patients treated by external drainage survived more than a short time, the immediate mortality being high; while on the other hand 5 patients in whom no operation was done survived. But among four cases of cholecystenterostomy (or anastomosis of the diverticulum with the intestine) there were three recoveries (Lavenson). So that it seems proper to advise that if the cyst cannot be extirpated (a dangerous and difficult operation), either it or the gall-bladder should be drained into the gastro-intestinal tract. In McConnell's patient, 500 cc. of bile were removed by extra-peritoneal puncture, after exposing the very large cyst by laparotomy; three years later the abdomen was reopened for recurrence of symptoms, and the cyst was found much smaller; nothing further was done. The patient, who suffered from cirrhosis of the liver and myxedema, was still living at the time of the report.

Courvoisier's Law.—In spite of enlargement of the ducts in common duct obstruction, the gall-bladder itself seldom is enlarged; as a rule it is contracted. This is in accord with what is known as

Courvoisier's Law[1] (1890) that in 80 per cent. of the cases of obstruction of the common duct due to stones there is contraction of the gall-bladder; while in 90 per cent. of the cases of enlargement of the gall-bladder the obstruction is due to causes other than stones.

Robson (1904) gave the following reasons for this condition:

1. All cases of cholelithiasis producing symptoms are accompanied by inflammation of the biliary passages, as shown by the almost universal presence of adhesions around the gall-bladder.

2. Gall-stones in the common duct seldom cause complete obstruction, either because they are floating in the duct or because they only partly fill it. There is, therefore, no sufficient backward pressure to cause dilatation of the gall-bladder.

3. The muscular coat of the gall-bladder contracts in efforts of explusion when there is any obstruction in the common duct.

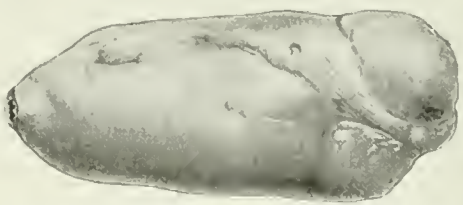


FIG. 140.—Extreme Contraction of Gall-bladder, the Result of Long-standing Cholecystitis. (Twice Natural Size.) From a Patient in the German Hospital.

4. The contraction, from being at first intermittent, becomes in the long run constant, and accompanying inflammation fixes the gall-bladder, which atrophies as a result.

The second of the above reasons given by Robson is considerably invalidated by the fact that the dilatation of the common duct itself and of the hepatic ducts demonstrates the existence of back pressure; while the fourth reason which is given as a corollary of the third, implies that the gall-bladder is in a condition of spasticity, which is far from being the case, as its muscular walls are destroyed by inflammatory infiltration long before this stage of cholelithiasis is reached. The explanation given by Courvoisier himself is sufficient to account for most cases: *that the longstanding inflammatory condition of the gall-bladder, whence in most instances the stones originated, has caused cicatricial contraction before the common duct becomes invaded.*

Violations of Courvoisier's Law.—Moynihan (1906) claims that

[1] Often known also as the Courvoisier-Terrier Law, because the phenomenon was pointed out independently by Prof. Terrier in 1891 before Courvoisier's monograph was known outside of Germany.

Courvoisier's law is violated (1) where there is a stone or stricture in the cystic duct causing hydrops or empyema, together with impaction of stone in the common duct; (2) where there is a stone in the cystic duct pressing upon the common duct; (3) where there is distention of the gall-bladder by an acute inflammatory process, with obstruction of the common duct by stone; (4) where there is chronic induration of the head of the pancreas with stone in the common duct; (5) where there is malignant disease of the common duct at any part of its course, or cancer of the head of the pancreas, and a chronic sclerosing cholecystitis.

The following case history illustrates the conditions most frequently found in cases of *obstruction of the common duct by calculus.*

L. B., female, aged thirty-one. Admitted to the German Hospital, January 16, 1907. Family history negative. Married thirteen years. Four pregnancies, two miscarriages. Bowels habitually constipated. Had typhoid fever eight years ago at which time she had marked pain in the gall-bladder region.

Present illness began in March, 1906, with severe colicky pains in the right hypochondrium, radiating to the back and right scapular region, and accompanied by vomiting. Confined to bed for one week, then up for a week and again confined to bed for eight weeks. Had constant pain in the gall-bladder region, with exacerbations of pain. Was able to be out of bed until September, 1906, during which period she did not feel well, had marked constipation, no appetite, and would perspire freely on exertion. In September was seized with excruciating pain in right hypochondrium, similar to first attack, but much more severe. Did not vomit. Had chill before onset of pain. Denies presence of jaundice in either attack. Following this attack was fairly well until two weeks before admission to hospital when she had attack of pain similar to the former attacks. Did not know she was jaundiced until told so. Great depression of spirits, appetite very poor. Bowels markedly constipated. Jaundice of skin and scleræ. Considerable tenderness over gall-bladder region. Lower border of liver palpable below costal margin. Abdomen soft except in the right hypochondrium where there is some rigidity. Hemoglobin, 80 per cent., W. B. C., 5900. Coagulation time, two minutes.

Operation, January 19, 1907. Ether anesthesia. Incision through upper right rectus. Adhesions between gall-bladder, duodenum, great omentum and transverse colon. Gall-bladder thickened, contracted and empty. All ducts thickened and surrounded by adhesions. Hepatic and common ducts greatly dilated. Common duct opened below junction of cystic and hepatic ducts. Probe passed into ampulla of Vater where a calculus was found. Calculus worked upward and removed through opening in duct. Rubber drainage introduced into common and hepatic ducts. Cystic artery and cystic duct ligated, gall-bladder removed. Gauze drainage along bed of gall-bladder and into subhepatic space. Wound closed to drainage with iodine gut. Jaundice disappeared three days after operation. Recovery uneventful. Culture of gall-bladder showed pure growth of Bacillus typhosus.

Enlargement of the liver generally follows obstruction of the common duct, the smaller biliary radicles becoming much dilated; in the course of time they may even produce a sort of varicose appearance on the surface of the liver. The back pressure eventually causes atrophy of the hepatic cells, and along with inflammatory changes due to infected bile, is responsible for the formation of new connective tissue in the substance of the liver, surrounding each biliary radicle. This is followed by *atrophy* of the parenchyma of the liver, so that there eventually is found a relatively increased proportion of connective tissue to the secreting parenchyma, and the liver becomes decreased in size. This condition closely resembles chronic cirrhosis, but Kelly (1908) distinguished between the present condition, which he termed chronic hepatitis, and true cirrhosis.

Calculi in the common duct frequently are productive of marked *changes in the pancreas*. These are considered in Chapter XXI. Extension of the common duct infection may occur by contiguity also to the *portal vein*, and rare cases are on record of portal and even splenic thrombosis, and of suppurative hepatitis due to embolism from these sources.

Calculi in the hepatic duct are rare. In the senior author's series of 549 cases of cholelithiasis (page 463), there was only one in which the stones were found in the hepatic duct alone; in twenty cases (3.6 per cent.) calculi in the hepatic ducts existed coincidently with stones in other locations, but with the one exception noted above in every case of hepatic duct stone there were also calculi in the common duct. In one other case not included in this series the senior author removed a single large stone from the hepatic duct. Courvoisier found that among fifty-nine cases of stones in the hepatic duct there were also stones in other locations in no less than fifty-six cases. These figures make it evident that in the vast majority of cases calculi in the hepatic duct have not originated in that situation but have either migrated first from the gall-bladder and cystic duct into the choledochus, and have thence floated up into the hepaticus; or, what is certainly much rarer, have formed in the choledochus or in the common hepatic duct as a result of calculous or other inflammatory obstruction of the lower portion of the choledochus.

The pathological changes induced by stones in the hepatic duct are not to be distinguished from those due to common duct lithiasis.

Predisposing Causes.—*Age.*—It is impossible to determine definitely at what age biliary calculi are formed, because no one can decide how long they may, or may not, remain quiescent after their formation. Between 50 and 60 per cent. of the cases that come to operation are over

forty years of age and less than 1 per cent. are under twenty. It is our belief, based upon clinical experience, that most gall-stones are formed between the ages of twenty and forty, when the system is most liable to the other infectious diseases such as typhoid fever, appendicitis, etc.

It is not at all unusual to note in the history that the "present illness" began five or ten years before admission to the hospital. If the time of the onset of the present illness were taken as the time of the formation of the gall-stones, it would be clearly demonstrated that cholelithiasis is a disease of early adult life and not of advanced age; and if it be remembered that even these first symptoms may not appear until years after the formation of the stone, this supposition is strengthened.

The above-mentioned opinion is at variance with the teachings of many of the older writers. Hoppe-Seyler states that "the increasing frequency of gall-stones with advancing years is very striking. Friedr. Hoffmann, Morgagni, Haller, Coe, J. P. Frank, and others have emphasized this fact, and it is established by all autopsy reports. As the latter alone furnish conclusive evidence, we are justified in believing that such a connection exists."

It is probable that these investigators were greatly influenced by the incidence of gall-stones at various ages. Necropsy reports are the only ones that are reliable in determining the prevalence of gall-stones at different periods of life; but, on the other hand, they are just as unreliable in determining the part age plays as an etiological factor in the formation of gall-stones, for the reason that biliary calculi may remain latent for years after their formation without giving rise to symptoms that will call attention to their presence. Hoppe-Seyler states that "in many, in fact the majority of the cases of concretions within the gall-bladder or the bile-passages, all symptoms are absent, and the condition is only discovered at autopsy. Whenever the stones remain in the location where they are formed, *i.e.*, in the gall-bladder, they usually produce no symptoms."

As has already been pointed out, catarrhal inflammation of the gall-bladder is believed to be one of the necessary factors in gall-stone formation; and the typhoid bacillus and the colon bacillus which are the organisms found most frequently in cholelithiasis, are most active in the human subject during the period between twenty and forty years of age, when typhoid fever, appendicitis, catarrhal inflammation of the intestines, etc., are most frequent. These two bacteria were found in 37.2 per cent. of the bacteriological examinations made by Kelly in patients with cholelithiasis operated upon by the

senior author at the German Hospital, while 55.4 per cent. of the cultures remained sterile (page 415).

There is no age at which gall-stones may not be found, John Thomson even claiming, according to Still (1899), that most of the gall-stones of early infancy are prenatal in origin, an infection of the biliary tract taking place during intrauterine life which in one instance will cause obliteration of the bile-ducts, in another the formation of stones. Still collected twenty-three cases of cholelithiasis in children under fourteen years, including three cases of his own; fourteen of the collected cases were under ten months of age; several were in still-born infants; his own three patients were each less than nine months old.

Sex.—Sex has a most decided influence on gall-stone formation. The clinical experience of the senior author places the preponderance of women over men nearly as four to one: of 606 cases of cholelithiasis 461 or 76.1 per cent. were females and 145, or 23.9 per cent. were males.

The greater frequency of gall-stone disease in women is due to conditions which favor stasis of bile in this sex. Chief among these is the habit of wearing tight corsets. Rother, according to Moynihan, found corset-liver in 40 per cent. of women suffering from gall-stones; and Schroeder (1892) found gall-stones in 59 per cent. of cases of corset-liver. But even when tight lacing is not carried to the degree necessary to produce a corset-liver limitation of the excursions of the diaphragm, caused by any constriction of the waist, interferes with the natural discharge of bile from the gall-bladder (page 37).

Pregnancy also is important as a predisposing factor in cholelithiasis; it has been shown (Payr) that during pregnancy the cholesterol content of the blood is increased. And the influence of pregnancy does not cease with the emptying of the uterus. While this organ is enlarged during the child-bearing period pressure is exerted on all the abdominal viscera, and the free evacuation of the gall-bladder is hindered by this pressure. After pregnancy terminates there often is a ptosis of the abdominal organs with twisting or kinking of the cystic duct, caused by traction, or at times, by torsion. Cholelithiasis seems to occur much more frequently in those women who have been pregnant, and especially in those who have borne many children, than in those whose uterus has never been gravid. Mayo (1911) says "90 per cent. of married women who have gall-stones have borne children, and 90 per cent. of these women identify the beginning of symptoms with some particular pregnancy."

The more frequent occurrence, in the female sex, of movable

kidney, especially of the right kidney, is also regarded as a predisposing cause of gall-stone formation, since by traction on the duodenum the bile-ducts may be displaced or kinked (page 521).

Climate.—From statistics that have been gathered from numerous sources by various investigators, it has been demonstrated that cholelithiasis is much more frequently seen in some localities than others. The cause of the greater frequency in these localities is not the climate or the altitude, however, but rather the different modes of living and the greater prevalence of infectious diseases which cause catarrhal conditions of the gastro-intestinal and biliary tracts.

Race.—This also seems to be a predisposing cause of cholelithiasis. Our clinical experience places the Hebrew race in the foreground in relation to the frequency with which gall-stones are found among the different races. Probably this is to be attributed to their sedentary habits. The African race, on the other hand, seems to be almost immune from gall-bladder affections.

Occupation.—Sedentary habits must be placed in this category. Statistics quoted by Douglas (1903) show that cholelithiasis is exceedingly common among teachers, clergymen and the insane. But this frequency is attributable not to the cerebral activity, but to the confinement and sedentary habits involved, which favor *stasis of bile.*

Cardiac lesions which cause stagnation of the bile may create a catarrhal condition of the mucous membrane, by venous stasis, or by rendering the patients sedentary in their habits, etc. Rolleston (1905) showed by the report of the Manchester Royal Infirmary, analyzed by Brockbank, that gall-stones are found twice as often in those patients who have cardiac lesions prior to their fatal illness as in those without such heart conditions.

The presence of any *foreign body in the biliary tract* may act as a predisposing cause of gall-stone formation, as it may become the nucleus of the subsequently formed calculus. Intestinal parasites, fruit kernels, silk threads, and other foreign bodies have been noted as forming nuclei of biliary concretions. Experiments performed by Mignot prove that foreign bodies may remain in the gall-bladder indefinitely if they are free from germ-life without causing inflammation or precipitation of the solids of the bile. There must be added an attenuated bacterial infection to cause the formation of calculi.

Any *septic condition*, especially in the abdominal cavity, predisposes to infections of the biliary tract, by acting as a focus from which bacteria may be carried to the bile-passages, through the systemic or the portal circulation. If the infection is attenuated when it reaches the

gall-bladder, gall-stone formation may result, if the other essentials are present.

Peritoneal adhesions, from any cause, will act as a predisposing cause of gall-stone formation by favoring stasis of the bile, through distortion or kinking of the bile ducts. In the same way, gastroptosis, coloptosis, cecum mobile, displacement of the right kidney, constipation, marked obesity, or any factor causing stasis of bile, may be regarded as a predisposing cause of cholelithiasis.

Degeneration of the *thyroid gland* and the *ovaries* is a predisposing factor in gall-stone formation, according to the views of Lorand (1906), who claims that as these glands govern oxidation and obesity, degenerative changes in them will cause atony of the biliary ducts, pathological alterations in the liver, atony of the intestine, constipation, etc., with resulting stagnation of bile.

Exciting Causes.—The exciting etiological factor in cholelithiasis used to be assigned to heredity, to cirrhosis of the liver, to gout, to rheumatism, to faulty metabolism, to errors and indiscretions in diet, to excessive use of alcohol, to digestive disturbances, etc. Modern science has removed all of these from the domain of exciting factors of cholelithiasis and has placed them either in the category of predisposing causes, or entirely without the realm of etiological factors, whether exciting or predisposing. There is but one exciting cause of gall-stone formation and that is an invasion of the biliary tract by attenuated bacterial infection in the presence of a stasis of bile.

A bacterial study of gall-stones shows the presence of bacteria in about 33 per cent. Such studies, however, are of no value in determining the action of bacteria as the underlying factor in gall-stone formation. It has been stated by numerous investigators and has been proved experimentally both in animals and in the test-tube, that gall-stones may rid themselves of bacteria after their formation; and, on the other hand, that biliary calculi may absorb bacteria from the surrounding bile. Thus it is quite possible to obtain colon bacilli from a gall-stone whose formation was due to typhoid infection, or *vice versa;* to obtain sterile cultures from biliary concretions universally acknowledged to owe their origin to bacteria, and to obtain cultures of bacteria which had nothing to do with the formation of the particular stone from which they were grown. No reliance should be placed on the results of bacterial investigation in the case of stones that have been examined months, perhaps years, after their formation. If, however, bacteriological examinations were to be made soon after the formation of the calculi, while they were still soft, and before the gall-bladder had had

an opportunity to rid itself of bacteria, micro-organisms would be found in every calculus with very few, if any, exceptions. According to Funke (1906) soft concretions invariably yield bacteria.

Symptomatology and Diagnosis.—The symptoms of cholelithiasis are presented in almost endless combination. A study of the pathological changes due to the presence of biliary calculi with added infection shows how this is possible, and will enable the surgeon so to interpret the signs and symptoms presented that, as a rule, not only a correct diagnosis of the presence of stones may be made, but also of their exact location and of the pathological lesions which will be disclosed by direct inspection of the affected part. It is essential, therefore, for anyone who attempts to diagnose and treat diseases of the liver and the biliary tracts to be thoroughly conversant with their pathology. Without such knowledge it will be impossible to interpret correctly the signs and symptoms presented and as a consequence the patient will suffer from faulty treatment. In the majority of cases of cholelithiasis the diagnosis is easy: symptoms and signs are presented which enable one to arrive readily at definite conclusions. In the minority of cases, however, it may be impossible to make a correct pathological diagnosis; and in some cases a diagnosis can be made only after the abdomen has been opened and the affected region has been examined by sight and touch.

If the conclusions of Kehr, with which Riedel agrees, are to be accepted, 95 per cent. of the patients possessing gall-stones do not present symptoms which direct attention to their presence. We cannot agree with these findings, although the opinions of such eminent clinicians must be given due weight. We fully agree that the majority of cases do not present the symptoms commonly recognized as classical in this condition; on the other hand, we believe that symptoms are present which usually are misunderstood. Such symptoms may be elicited by studying carefully the history of the case. Notes of former illness, of "biliousness," of attacks of "indigestion," etc., should be made fully and in minute details. Inquiry regarding attacks of pain, no matter how remote, should be made, and the replies noted. The presence or absence of nausea or vomiting, of flatulence, or constipation, of fever, of chills, or of jaundice during any period in the past history should be carefully noted. Negative findings often are as valuable as are positive, in arriving at a correct diagnosis.

Many symptoms of cholelithiasis which often are classified vaguely as stomach and liver troubles, "biliousness," "indigestion," "dyspepsia," etc., will be found in such a history, and will often lead to the

diagnosis of biliary calculi before any marked symptoms have presented themselves. Riedel (1903) claimed that "cramps" in the stomach are caused by gall-stones in ninety-seven out of 100 cases. When the so-called classical symptoms are present there is little, if any difficulty in making the diagnosis.

Gall-stones, unaided by infection, present scarcely any symptoms. All signs and symptoms presented during the course of gall-stone disease, are due, we believe, to inflammation of the biliary tract resulting from infection. The symptoms vary with the severity and chronicity of the infection.

Pain is the most constant symptom of cholelithiasis and one of the most important to be studied in making a diagnosis. It is caused in all cases, we believe, by infection or the results of infection of the biliary tract and never is due to the mere presence of the gall-stones. In a series of 549 cases operated upon by the senior author at the German Hospital, pain was present in 539 or 98.2 per cent., and was absent or not obtainable in the history of only ten, or 1.8 per cent.

It is convenient to discuss the symptoms of pain in cases of cholelithiasis under the headings of *local* and *referred* pain, a plan followed by Moynihan.

Local pain, which will be considered first, is met with in two main types, the first embracing what are now generally recognized as the "gastric symptoms" of cholelithiasis, and the second being the classical "biliary colic." The "gastric symptoms" of cholelithiasis frequently are followed by symptoms of acute calculous cholecystitis without the patient ever experiencing an attack of biliary colic. (The symptoms of acute cholecystitis have been considered at page 454.)

Gastric Symptoms.—The earliest symptom generally is pain of a character and location that is very misleading to the patient and to many medical observers. The patient attributes this initial pain to a bilious attack, to gastralgia, to dyspepsia, to neuritis, to gastritis, to indigestion, in fact, to anything except to gall-stones. It is described by the patient as dull, burning, gnawing, boring, grasping, etc. As a rule it is confined to the epigastrium. These "gastric symptoms" have been described as prodromal symptoms which "are said to indicate the impending formation of gall-stones" (Hoppe-Seyler). Later study has shown them to be symptoms caused by gall-stones confined to a gall-bladder in which there is very mild acute or chronic catarrhal inflammation, the latter affecting the gall-bladder alone, while the cystic duct is occluded momentarily if at all, and the to-and-fro motion of the bile is practically normal. It is recognized as gall-stone pain by the irregu-

larity of its occurrence and by its dependence on no recognized factor. It may occur at night or during the day and be independent of the ingestion of food or of the kind of food. As Graham says "these are light attacks of distress, gas, upward pressure, coming often soon after food or at irregular times, often of sudden onset, short duration, eased by belching or perhaps slight vomiting, regurgitation, or slipping away almost unnoticed and without treatment." No particular effect is produced on the patient's constitution, and good nutrition is maintained. This initial pain is never of a colicky character.

It was pointed out at page 142 that pylorospasm, gastro-succorrhea, etc., are not infrequently due to cholelithiasis; and we have repeatedly called attention to the fact that gastric lesions are often secondary to infections of the biliary tract.

There may be slight tenderness on pressure over the gall-bladder (see page 487), but more moderate pressure in this region will relieve whatever pain is present. Some writers have noted a heavy dragging pain or sensation due to the weight of the secretions within the gall-bladder. Our clinical experience has failed to confirm such findings in any case. When this symptom occurs, some pathological lesion other than biliary concretions within the gall-bladder will be present to account for it. Pericholecystic adhesions, carcinoma or other complications will be found, and it is in such cases that referred pain occurs (page 485).

With more active inflammation of the gall-bladder, the symptoms presented are, first, those of cholecystitis (page 454); later there occur the symptoms of the complications that arise during the inflammation, or those of the sequels that follow its subsidence. As has already been pointed out a persistent chronic cholecystitis usually remains after gall-stone formation; and this chronic condition is liable to be interrupted by attacks of inflammation more or less acute in type.

Biliary Colic.—The typical "*biliary colic*" of the text-books is present in a great majority of cases that are operated upon, but it is not to be found in all of them. In the previously mentioned series of 549 cases of cholelithiasis, pain of a distinctly colicky character was noted in 462 or 82.3 per cent.; and was unrecognized by the patient at any time in ninety-seven or 17.6 per cent. This colicky pain is caused by muscular contraction of the gall-bladder or ducts, and is not due directly to the movements of a stone either in the gall-bladder or through the ducts, although movements of the stones may add to the severity of the colic. It is well known that in exceptional cases biliary calculi are recovered from the feces of patients who have never suffered from

"biliary colic;" and that attacks of biliary colic are very seldom followed by the passage of a calculus; while every surgeon of experience has operated on patients who have never passed any stones in spite of numerous attacks of colic, and yet in whom no gall-stones were to be found at the time of the operation.

Biliary colic, in other words, is the equivalent of renal colic, appendicular colic, and intestinal colic: in all varieties the pain is not primarily caused by the passage of a foreign body, but by disordered and violent peristaltic action of the diseased organ, which has been excited in the effort to overcome an obstacle to its evacuation. This obstacle is rarely a foreign body in the nature of a calculus; almost invariably it is due to some other form of obstruction, either stenosis of the lumen from acute inflammatory edema, to kinks produced by displacement of the organ affected, or to viscosity of its secretion. There is distention of the gall-bladder or ducts with bile and mucus, and an attempt to force a passageway through the duct. If an obstruction in the cystic duct is relieved by a stone returning to the body of the bladder or by the subsidence of the inflammatory obstruction at the mouth of the duct, the pain disappears at once, the gall-bladder emptying itself through the duct. In the mildest cases the pain may be fleeting; of such short duration, in fact, that the patient will soon forget that he has experienced it and can recall it only after the most thorough questioning. This acute, initial attack of mild colic causes no pain, as a rule, in the region of the gall-bladder—the pain is in the mid-epigastric region. In other cases, however, the initial attack is severe, sudden, overwhelming. A man, believing himself to be in the enjoyment of perfect health, except for slight gastric symptoms which have never seriously annoyed him, may suddenly have a dreadful cramp in his upper abdomen; he bends forward, pressing his hands or the back of a chair into his belly; breaks out in a cold sweat; becomes deathly pale and feels faint; is nauseated; and sometimes his distress is relieved by vomiting. At other times he will writhe around his bed, or even on the floor, in utmost agony. Hot applications to the epigastrium or hypochondrium may have scarcely any effect on the pain, and even morphin hypodermically does not always bring relief as soon as could be wished. Death sometimes occurs during the paroxysm (Langebuch, 1897). The fever which follows the sweat may rise to 104° F., or higher.

When the obstruction is not relieved the pain will be more lasting, remaining for hours or even days, not so intense as at first, but still severe. The pain now passes to the gall-bladder region, with pain

referred to the back and shoulder. This pain is significant because it usually indicates that there are taking place, in the neck of the gall-bladder or in the cystic duct, changes which will result in permanent damage to these structures. The obstruction may be relieved by a stone passing through the cystic duct, or by the subsidence of the acute inflammation of the duct, but the lesions will be permanent and the parts cannot again return to a normal condition. Simple hydrops, empyema, gangrene, or perforation may result.

With complete obstruction of the cystic duct resulting in simple hydrops, colicky pain usually disappears; with incomplete obstruction the colic continues indefinitely. In the latter class of cases, there often is a referred pain in the right iliac fossa, at times paroxysmal and sharp, resembling in many respects the pain found in connection with an impacted ureteral calculus.

When the inflammation involves the common duct, or when a stone in its passage has reached this location, the colicky pain will be felt in the right hypochondrium, usually associated with referred pain in the right shoulder. Subsidence of the inflammation will be followed by cessation of pain, even if a gall-stone remains in the duct.

Yet even during the intervals between attacks or in the cases of patients who have had no distinct attacks of biliary colic, there is seldom a feeling of perfect comfort in the gall-bladder region. The symptoms of indigestion, of gastric flatulence, etc., after meals, will persist, perhaps becoming more pronounced; the clothes will be loosened around the waist; and moderate pressure or support with the hand to the right hypochondrium will be attempted to relieve the dragging sensation usually significant of pericholecystic adhesions, and chronic cholelithiasis. Langenbuch, and later Moynihan, have called particular attention to a sensation of chilliness, during the evening, as particularly suggestive of mild infection of the gall-bladder.

Referred Pain.—As pointed out at page 26 the various positions to which pain caused by affections of the liver and biliary track may be referred can readily be explained by a knowledge of the nerve supply of the organs involved. The liver and biliary tract are supplied by three nervous systems, the *cranial*, the *spinal*, and the *sympathetic*. These connections have been well summarized by Millard (1907).

The tenth *cranial*, or pneumogastric nerve, distributes sensory fibres to the esophagus, the stomach, the lungs, and special fibres to the heart, liver, spleen, pancreas, kidneys, suprarenal bodies and the intestinal blood-vessels. The *spinal* system is represented by the phrenic nerve which is derived mainly from the fourth cervical nerve, a

branch of which, the supraacromial, is distributed to the integument of the point of the shoulder. The phrenic distributes fibres to the diaphragm and falciform ligament of the liver; it unites with filaments of the celiac plexus to form the diaphragmatic plexus which is joined by filaments from the diaphragmatic ganglion. From this plexus fibres are distributed to the coronary ligaments and peritoneum of the liver and to the right suprarenal. The *sympathetic* nerve supply is derived from the celiac plexus, which is joined by branches from both semilunar ganglia and from the right pneumogastric. From it arise the coronary, hepatic, and splenic plexuses. The hepatic plexus inosculates with fibres from the left pneumogastric and enters the liver. Filaments are distributed to the right suprarenal; other filaments follow the branches of the hepatic artery.

On account of this general intercommunication of the nerve supply of the liver, biliary apparatus and other regions of the body, pain caused by lesions of these organs may be referred to various positions. It may be purely epigastric; may occur in the right or left hypochondrium; in either kidney region; in the diaphragmatic area, in the cardiac region, or in the left lung; in the back; beneath either shoulder blade; at the tip of the shoulder; or throughout the abdomen.

In the earliest stages of cholelithiasis, where the gastric symptoms predominate, the pain rarely is referred. Sometimes a dull aching, a feeling of discomfort, is experienced to the right of the spinal column. When the disease has lasted for some time, and pericholecystic adhesions have developed, implicating the pylorus, the pain may be referred to the left subscapular region (p. 4).

In attacks of biliary colic the pain is very constantly referred, either to the right shoulder, to the right chest, or more rarely to the right groin. In colic from involvement of the common bile-duct the entire lower right thorax may be painful, from swelling of the liver.

The character, location, and duration of pain are important diagnostic factors both in the diagnosis of infections of the biliary tract and in the differential diagnosis of all other lesions in the upper portion of the abdomen. The peculiarities of the pain should be carefully considered whenever present; and if absent this fact should always be noted.

Pain in "gastric symptom cases" of cholelithiasis is dull, burning, gnawing, boring, or grasping. It is confined to the epigastrium or possibly referred to the back. It is independent of the ingestion of food or of the kind of food. It may occur at night or during the day.

The pain of more severe infection of the biliary tract is colicky in character, very severe, of sudden onset, usually of short duration, and of sudden cessation. It is felt in the epigastrium or the right hypochondrium and may be referred to the breast, back, right costal arch, right shoulder, shoulder blades, etc. The attacks are independent of the ingestion of food and usually of the kind of food. They may occur at night or on an empty stomach. The intervals between attacks may vary from hours or days to years.

The pain in *ulcer of the stomach* usually is of a stabbing character, located in the epigastrium, being referred at times to the left hypochondrium or left scapular region. It is rarely present when the stomach is empty, but follows the ingestion of food either immediately or within half an hour, and often increases in severity until the stomach has been emptied by vomiting or by the expulsion of stomach contents into the duodenum. The pain is seldom absent for any long periods of time.

The pain in *ulcer of the duodenum* usually recurs during definite periods of time, the duration of the periods varying from a few days to several months. During these periods, the pains will appear daily or several times a day. They are burning and gnawing in character, and radiate to the region of the stomach and duodenum. They bear a regular relation to the patient's meals: they are at their height from two to six hours after the ingestion of food, and are relieved or entirely dissipated by food or drink or by removal of the contents of the stomach either by vomiting or by irrigation. Between these periods the patient considers himself in perfect health; whereas in cholelithiasis a certain amount of gastric distress is constantly present even during the intervals of freedom from acute symptoms.

The pain in *cancer of the stomach* is more or less continuous, of a dull, depressing character. It is generally increased by the ingestion of food.

Tenderness obtained by pressure or palpation is closely allied to pain in all cases of cholelithiasis. Some writers lay much greater stress on certain points of tenderness or pain elicited by pressure, than on any other symptom. This tenderness is claimed by some to indicate, absolutely, by its presence or absence, the presence or absence of gall-stones. Robson and Cammidge (1909) state that "another characteristic symptom of great diagnostic value is the existence of a tender spot an inch above the umbilicus, and in a line between it and the right costal margin. This tender spot is quite as constant as the McBurney point in appendicits, although in some

cases it may be a little higher than that mentioned, but in the same line." This is frequently spoken of as "Robson's point."

This characteristic point of tenderness may also be elicited in the following manner, usually called the Murphy method. With the patient sitting up and leaning forward, the examiner stands back of the patient with one hand hooked under each costal margin at the ninth costal cartilage. Deep inspiration forces the liver and gall-bladder downward while the finger-tips press inward and upward. In this way pressure on the kidney is avoided, while tenderness from the stomach or duodeum is much more superficial. Pain is experienced

FIG. 141.—Palpation of the Gall-bladder by "Thumb Pressure" under the Ninth Costal Cartilage.

by the patient as pressure is exerted on the deep-lying gall-bladder and the inspiration is checked suddenly by the pain, the patient being unable to take a full inspiration. While we believe that this tenderness is very significant of the presence of biliary calculi, it is scarcely warrantable to go as far as Bishop (1907), who said that "if the result of such examination is negative, the possibility of biliary calculi is eliminated." Monsarrat (1908) claimed that this tenderness is the one persistent clinical sign upon which a diagnosis may be made. This tender point is also elicited by "thumb pressure" under the ninth costal cartilage, after the manner practised by Moynihan.

The left hand is placed over the lower right thorax as the surgeon sits or kneels at the right side of the patient's bed, and the thumb of this hand presses firmly but gently over the region of the gall-bladder just below the costal border. Or the surgeon may employ the method which, according to Langenbuch, was originally recommended by Rheinstein (1891): The left hand is placed under the patient so as to steady the lower right thorax; then by bimanual palpation, using the right hand over the upper abdomen, the two hands are gently but firmly approximated, and as the right kidney

FIG. 142.— Bimanual Palpation of the Gall-bladder.

and the liver are pushed forward, the tenderness due to the diseased gall-bladder becomes evident at the end of deep inspiration (Figs. 141, 142).

No undue force should be used in palpating the gall-bladder region, for rupture of a diseased gall-bladder has been recorded (Langenbuch) as due to such manœuvres.

There also is very constantly a tender spot in Boas's area, on the right side behind, at the level of the twelfth dorsal vertebra, two or three finger breadths from the spine, and this may be present even when no tenderness can be discovered in front.

While these tender spots are of significance when present, their absence does not preclude the presence of gall-stones. Our clinical

experience has been unable to confirm the constancy of these diagnostic signs as observed by others. They have been absent or not obtainable in a number of cases, especially in some "gastric symptom cases" where the gall-stones and the inflammation have been confined to the inside of the gall-bladder. Where there is or has been pericholecystic inflammation, tenderness is much more constant. In such cases slight rigidity of the over-lying muscles is present, while in cases of cholelcystitis, and other acute phases of gall-bladder disease, both tenderness and rigidity are pronounced.

Enlargement of the Gall-bladder.—The gall-bladder is enlarged in the majority of cases of cholelithiasis, but the enlargement may not be sufficient to cause the presence of a palpable tumor, on account of the position of the gall-bladder in its fossa on the under surface of the right lobe of the liver. The normal gall-bladder cannot be felt through the abdominal wall; when the gall-bladder is palpable it must be distended sufficiently to force its fundus beyond the border of the liver. It is possible for the gall-bladder to be very tense without there being a palpable tumor, since the gall-bladder wall may be thickened and sclerotic from long-standing disease, and be incapable of further distention; thus is it not unusual to find a small contracted gall-bladder full of pus (empyema), and very tense. In a series of 111 cases of cholelithiasis operated upon by the senior author, the gall-bladder was distended in seventy-four, or 66.6 per cent.; it was normal in size in seven, or 6.3 per cent.; and was contracted in thirty, or 27 per cent.

The position of the gall-bladder varies with the size and position of the liver; in normal conditions, the neck of the gall-bladder is about on a level with the ninth costal cartilage. In cases of ptosis of the liver or enlargement of that organ, the neck of the gall-bladder may be as low as the umbilicus or even below that point. If the gall-bladder is enlarged the fundus usually moves downward and forward, although it is not rare to find the tumor extending toward the median line, nor is it unusual to find at operation that the gall-bladder, though enlarged, is hidden behind the liver and therefore was not palpable through the abdominal walls. In some instances it has extended to the brim of the pelvis, and in this position has been mistaken for an ovarian tumor (page 451).

When the distention of the gall-bladder is sufficient to form a palpable tumor, the latter will be felt as a rounded, smooth pear-shaped mass, readily movable laterally in the absence of adhesions. In the presence of pericholecystic adhesions, the tumor may not be

recognizable as the gall-bladder, the characteristic shape and smoothness being absent. Under these conditions a mass will be palpable, more or less fixed. The mass may move with respiration or be fixed to the anterior abdominal wall. When the gall-bladder is free there is a distinct movement with respiration, the dome of the tumor passing under the palpating hand with each respiratory movement.

In the more severe infections it is often impossible to determine the presence of a tumor, even though it may be very evident at operation, on account of the rigidity of the overlying muscles. There must be more or less laxity of the abdominal muscles in the right upper quadrant, if the gall-bladder is to be palpated.

The enlarged gall-bladder, unless held in place by adhesions, may be moved or pushed from its normal location, but will return immediately when relieved.

A tumor of the gall-bladder without jaundice usually indicates closure of the cystic duct, generally by a calculus.

A tumor of the gall-bladder with jaundice, is indicative of pressure from without, in about 90 per cent. of such cases. Jaundice due to calculus obstruction usually is accompanied by a contracted gall-bladder (see page 473). Enlargement of the gall-bladder with jaundice usually is significant of carcinoma of the head of the pancreas, of the duodenum, or of the common duct; or of benign disease of the pancreas.

Fever.—An increase of temperature at time of operation for cholelithiasis is noted in about one-third of the cases. In a series of 368 cases of cholelithiasis operated upon by the senior author, fever was absent in 264, or 68.4 per cent.; and was present in 122 or 31.6 per cent. The rise in temperature is due to an infectious process, and naturally is absent except during the activity of the infection. With the subsidence of the acute infection, the fever disappears. So long as the infection is confined to the gall-bladder there rarely is any marked rise in temperature, since the gall-bladder is nearly devoid of lymph nodes and very little absorption occurs from it. But when the infection spreads either to the surrounding peritoneum or to the bile-ducts, then marked temperature changes occur. The temperature in cases of cholangeitis, biliary hepatitis, etc., may resemble that of malaria or or septicemia. In such cases the sudden onset of fever, its rapid rise to a height if 101° F., or more, and its quick fall again to the normal, are quite characteristic, the temperature forming what Moynihan has well named a "steeple" chart. There is entire absence of fever between the excerbations.

Vomiting.—Vomiting usually is present during the attacks of biliary colic, and usually is absent between the attacks. At times it begins with the termination of the attack of colic; first the ingested food and later bilious matter being rejected. Blood is very rarely present. The vomiting often seems to relieve the pain. In rare instances the vomiting may be persistent, even alarming.

Vomiting in *ulcer of the stomach* is almost as constant as is pain in that affection. Vomiting usually brings absolute relief. The vomitus is sour, often contains an excessive amount of liquids, and is streaked with blood in over 25 per cent. of the cases.

Vomiting in *ulcer of the duodenum* usually increases in severity with the progress of the disease, at times becoming alarming. It usually occurs about two to four hours after the ingestion of food, at the time the pain and gas are greatest. The vomitus is "acid," "acrid," "bitter-burning," rather small in amount but very irritating. It sometimes contains blood.

Vomiting in *cancer of the stomach* is irregular. Some relief is generally afforded. The vomitus is foul-smelling and consists of partly digested food and contains altered blood in about two-thirds of the cases.

Crepitation.—In a few instances a strong crepitation may be elicited by pressure on a gall-bladder containing calculi. LeBlanc (1906) reported such a case, in which auscultation also elicited a sound exactly similar to the crepitus of a fractured bone. Such cases are rare.

Tetany has been referred to at page 158 as frequently of gastro-intestinal origin. It is occasionally observed on the subsidence of a severe attack of biliary colic.

Roentgenography, for a long time considered a very unreliable aid in the detection of biliary calculi, has been so developed in recent years that it must be regarded almost as indispensable as in the diagnosis of kidney stones. But it must be remembered that even in the absence of stones, the biliary tract may be very seriously diseased, and a negative X-ray examination should not be held to contraindicate exploratory operation, when the history and clinical symptoms indicate this to be proper.

Localization of Gall-stones.—In most cases of cholelithiasis it is possible to ascertain with a fair degree of accuracy whether the calculi still remain in the gall-bladder or whether they have escaped into the bile-ducts, and if so whether the common duct is involved. As calculi may be found in several ducts as well as in the gall-bladder at the same time, the clinical picture may be somewhat confusing; but a distinction

between stones still in the gall-bladder, those in the cystic duct, and those in the common duct, usually can be made by attention to the history of the case and a careful physical examination.

The symptoms and signs of **stones in the gall-bladder** alone ("simple cholelithiasis" as it is called) are those of chronic catarrhal cholecystitis. There may be an occasional exacerbation resulting in acute cholecystitis, or in gall-stone colic; and it is chiefly on the *recurrence of symptoms* that the diagnosis of gall-stones is based. A single attack of cholecystitis, even with severe colic, is not enough to warrant a diagnosis of stone.

Stone in the Cystic Duct.— The frequency of this localization of gall-stones is shown in tabular form at page 463. Including cases where calculi are found elsewhere in the bile-tract as well as in the cystic duct, this portion of the tract is involved in about 20 per cent. of cases. The symptoms and signs vary with the size of the stone, the acuteness of the inflammation, and the completeness of the obstruction. As soon as a stone impinges upon the mouth of the duct or enters its lumen, typical gall-stone colic results from the attempt to rid the duct of the obstruction. Paroxysms of pain recur until the stone either passes through the duct, returns to the gall-bladder, or becomes lodged permanently in the duct. In the latter case obstruction is not necessarily complete, since usually a portion of the lumen remains through which bile can pass. Should the obstruction become complete, however, the pain will disappear as further atempts at dislodgment gradually cease. The pain of obstruction of the neck of the gall-bladder is referred to the right shoulder-blade in 70 per cent., to the left scapular region in 10 per cent., and to the sternum, precordial region, and right subclavicular region in 20 per cent. of cases, according to McBurney (1907).

Jaundice is not present in uncomplicated cases of calculus in the cystic duct. The presence of jaundice implies cholangeitis and obstruction of the common or hepatic duct.

The presence or absence of *enlargement of the gall-bladder* depends upon the character and degree of inflammation in the organ before the stone enters the duct, and upon the completeness of the obstruction. When there has been previous inflammation, the gall-bladder walls usually are thickened and contracted; or the entire organ may be held fast by surrounding adhesions. In these cases no enlargement of the gall-bladder will occur. In the majority of instances, however, a dilatable gall-bladder is present, enlargement occurs and the gall-bladder can be palpated. If obstruction of the duct is complete, no

bile can enter, and the gall-bladder becomes distended with its secretion, forming *hydrops vesicæ felleæ*, the symptoms of which were considered at p. 455. Should acute infection occur in the gall-bladder empyema develops. The symptoms of this condition were discussed at page 456.

The *diagnosis* of obstruction of the cystic duct depends upon a correct interpretation of the symptoms enumerated above, and on the recognition of enlargement of the gall-bladder. When the gall-bladder is small or contracted or bound down by adhesions it is nearly impossible to make a diagnosis of obstruction of the cystic duct. Moreover, enlargement of the gall-bladder may be caused by any condition which causes obstruction to the flow of bile, or even by the presence of a large number of calculi within the gall-bladder.

Pressure on other structures exerted by a stone in the cystic duct may give rise to symptoms that will make the diagnosis more difficult. Thus cholangeitis and, at times, obstructive jaundice may result from pressure on the common duct; thrombosis and ascites may result from pressure on the portal vein; symptoms of gastric dilatation may result from pressure on the duodenum. But these conditions are comparatively rare, and the history of the case should aid in clearing up the diagnosis, especially when enlargement of the gall-bladder is present.

The *prognosis* in obstruction of the cystic duct is good, unless the contents of the gall-bladder become infected so virulently that acute empyema, perforation or gangrene results. The prognosis under such conditions is very grave. In uncomplicated cases the operative mortality is no higher than that of cholecystectomy in general.

Stone in the Common Duct.—The frequency of stone in the common duct was discussed at page 463. Including cases where calculi are found elsewhere as well as in the common duct, this is involved in over 21 per cent. of the cases. The more experienced a surgeon becomes, the less apt is he to let a stone in the common duct remain undetected at operation.

The *symptoms* presented are pain of a colicky nature, followed in most instances by jaundice which is intermittent and which varies greatly in intensity even when present. The attacks of pain often are associated with intermittent feverish attacks, and with chills; and in most instances such attacks are followed by an increase in intensity of the jaundice. The chain of symptoms presented is similar to that of "Charcot's Intermittent Fever," a type of cholangeitis associated

with obstruction of the common duct. The symptoms of cholangeitis have been described at page 448.

There is no enlargement of the gall-bladder in uncomplicated cases of obstruction of the common duct by calculus. There are few exceptions to this rule; they have been discussed at page 474.

The *diagnosis* of calculous obstruction of the common duct depends largely upon the history of the case. If the patient is seen during an attack of acute cholangeitis it may be possible to make a diagnosis even in the absence of an accurate history. Obstruction of the common duct by calculus may be differentiated from that due to carcinoma of the duct by the train of symtoms usually noted in the latter affection. Chronic progressive jaundice, without fever, with decoloration of the stools, absence of sharp pain, moderate enlargement of the liver, a distended gall-bladder, and continuous emaciation, are characteristics of obstruction from cancer of the common duct (page 588). Obstruction of the common duct from enlargement of the head of the pancreas, as a result either of inflammation or of a new growth, is considered at page 695.

The *prognosis* of calculous obstruction of the common duct is very much graver that where the cystic duct alone is obstructed. The danger is in large part due to the more serious lesions present, but in no small degree to the more extensive operative interference required to assure removal of all calculi. The mortality of choledochotomy varies from 7 to 30 per cent., in various statistics.

Treatment of Cholelithiasis

After the discussion of the pathology of this affection, into which we have gone at some length, it scarcely seems necessary to insist further on the fact that cholelithiasis is a surgical disease, and that operative treatment is requisite for its cure. Kocher's oft-quoted epigram that gall-stones belong primarily not to the surgeon but to the patient, is true enough; but the modest statement of fact which he appends should not be overlooked. It is to the effect that it is the patient's privilege, if he so elects, to spend an invalid's life sojourning from time to time at the various resorts such as Carlsbad, Marienbad, Saratoga, etc., thus cautiously endeavoring to keep his calculi quietly at rest in his gall-bladder; or even to endure with fortitude the occasional agony induced when a calculus leaves the gall-bladder and attempts to force its passage into the bowel. But this life is only for the well-to-do classes; as Kehr says, at Carlsbad the poor disappear abso-

lutely; "the patients come to Carlsbad usually in the period of latency; the minority still have colics or inflammation of the gall-bladder. Now begins the regular living, the beneficial, pain-assuaging, laxative action of the Carlsbad springs, the delightful influence of the Sprudel baths with their peat poultices to the liver and region of the gall-bladder. The beautiful surroundings" he proceeds "entice the cure-guest into the noble forest, he climbs the mountains, which in stillness leave nothing to wish for, and he forgets the worry of his business and the pain of his disease. The cuisine permitted by the cure removes the sins of his club life at home, of the many strawberry and peach punches; briefly, the tissue changes are powerfully stimulated, and whoever is not very sick must in a very short time indeed feel himself well." But while such a life is a patient's privilege if he can afford it, there are very many who cannot, and it is the duty of the attending physician to inform his patients that modern surgery offers a rapid and a lasting cure at a very trifling risk, provided operation is undertaken before complications have arisen. The best time for a Carlsbad "cure" is after operation.

In uncomplicated cases the mortality of operation is less than 5 per cent., and in the hands of those who have much experience in this work it has been reduced as low as 2 or even 1 per cent. When complications develop, the mortality rises rapidly, as will be noted on subsequent pages. Not only is the migration of calculi from the gall-bladder into the duct a factor of very serious moment in increasing the death-rate from cholelithiasis, but even when the stones remain in the gall-bladder the occurrence of acute cholecystitis or its possible sequels, such as pericholecystic adhesions, perforation or gangrene, may put the patient's life in jeopardy at any time. The safest course, by far, for the patient to pursue is to have his gall-stones removed as soon as they begin to produce noticeable symptoms. At this time the removal of the calculi with the simple operation of drainage of the gall-bladder, or of cholecystectomy, will effect a cure. Later not only will cholecystectomy be necessary, and a more serious undertaking than in the early stages of disease, but incision and drainage of the common duct or even of the hepatic duct may be required. These operations, and others yet more complicated, have a much higher mortality, and often gravely tax the technical skill as well as the judgment of the surgeon. We believe there is no more difficult surgery than that of the bile-ducts.

The comparative mortality of operations for these various conditions may be seen in the following table:

OPERATIONS FOR CHOLELITHIASIS (1912–1920)
(Statistics from the Lankenau Hospital)

Lesions	Operations	Deaths	Mortality per cent.
Without Acute infection[1]	628	42	6.7
With: Acute cholecystitis	37	2	5.6
Empyema	23	1	4.3
Hydrops	2	0	
Gangrene or perforation	7	3	43.0
Pericholecystic abscess	1	0	
Acute pancreatitis	8	3	37.5
Acute cholangeitis	3	2	66.0
Chronic pancreatitis	46	2	4.3
Pancreatic lymphangeitis	40	1	2.5
Ulcer, duodenal	7	2	28.5
Ulcer, gastric	2	0	
	804	58	7.0

[1] Including calculi in the common and hepatic ducts.

Recurrence of gall-stones after operation is exceedingly rare. Although, as noted at page 423, calculi occasionally have formed around a silk suture or other foreign material left in the gall-bladder at operation, usually "recurrence" implies not a new formation of stones, but that some calculi were overlooked at the time of operation. If operation is undertaken at the propitious time, that is, before any calculi have left the gall-bladder, and before pericholecystic adhesions have developed as the result of attacks of cholecystitis, then the complete removal of the calculi is not difficult, and no stones should be overlooked. This is a great argument in favor of early operation. Maurice Richardson reported a case in which, after dislodging a stone from the common duct, it escaped into the hepatic duct; and he found himself absolutely impotent to find it again: "there was the patient with his common duct wide open, and somewhere in the depths of the liver was the offending stone, ready to be drifted down into the same impaction as before." Fortunately such a catastrophe seldom occurs, even to surgeons of less skill and experience than Richardson.

Robson calls attention to the fact that in the very large experience of Mayo, Kehr, and himself, it had been stated by each separately that the recurrence of gall-stones after cholecystostomy was an extremely rare event. In 1911 W. J. Mayo stated that among 4000 operations performed upon the biliary tract by himself and C. H. Mayo

only three cases were observed in which stones had reformed in the gall-bladder.

But while this freedom from recurrence is the rule, a candid statement of fact must be made that very occasionally true recurrences of gall-stones (not cases of overlooked stones) do occur. We have encountered a few such cases ourselves. The following history showing that not very many months after the senior author had removed 100 calculi from a patient's gall-bladder another surgeon removed over 200 stones from it, is incontrovertible proof of recurrence. Two hundred calculi could not have been overlooked at the first operation.

Miss——, nurse, operated on at the German Hospital in 1897 for calculous cholecystitis, removal of 100 gall-stones. Remained perfectly well for one year when she had a recurrence of cholecystitis and subsequent operation with removal of 200 stones.

The thing which demands elucidation is not why recurrences occur, but why they do not occur more often. True, these patients when operated upon have passed the age during which infectious diseases such as typhoid fever, entero-colitis, etc., are prone to occur. When they are relieved of their calculi and the existing infection of the bile-tract is cured by drainage it may be argued that no renewed infection of the biliary tract is likely to develop, because these predisposing causes of biliary infection will not again arise. Yet such renewed infections occasionally do occur. They are extremely rare, however, and would be rarer still we believe were cholecystectomy more often employed in cases of simple cholelithiasis; or, if the gall-bladder is not removed, were postoperative drainage of the gall-bladder maintained for a longer time than often is the case.

But though early operation is the best treatment, in cases of *simple cholelithiasis* it may be very difficult to persuade the patient to submit to operation. If he has had no acute attacks, either of cholecystitis or gall-stone colic, and if he suffers merely from symptoms of "indigestion," he may be quite satisfied to stay as he is; and a surgeon must feel very sure of his ground before attempting to convince such a patient against his will. Of course one must bear in mind the slight but seemingly unavoidable mortality which attends any large series of operations. An occasional death will occur from pneumonia, following the anesthetic. But as Kehr says, "*the slight dangers of early operation stand in no sort of a relation with the great dangers of the disease itself*. . . . . .Even the latent cholelithiasis we should always regard with suspicious eyes, for the quiet

work of gall-stones is often the most destructive." Carcinoma may develop from stones which cause no particular distress; and perforations into hollow viscera (internal biliary fistulæ, page 525) often occur without producing any acute symptoms. "The danger of carcinoma alone," writes Mayo, "is five times as great as is the mortality following operations for the relief of simple gall-stone disease." "*In malignancy and insidiousness,*" concludes Kehr, "*no disease of man compares with cholelithiasis.*"

Yet operation is not to be insisted upon indiscriminately in every case. The *contra-indications to operation* may be summarized as serious organic lesions of the heart, lungs, or kidneys; extreme age; anemia and slow coagulability of the blood. Such patients as these must lead an invalid's life and may hope by careful dieting and attention to hygiene to prevent the development of acute complications which will render operation imperative at all hazards.

What the type of operation shall be in these interval cases is a subject that has been discussed in recent years a little too strenuously. Certain surgeons contend that in every case the gall-bladder should be removed. They regard it as a hotbed of infection, and claim that its retention favors reformation of calculi. They dwell upon the supposed functional uselessness of the gall-bladder, and upon its pathological importance. They magnify the frequency of persistent fistulæ, or reformation of calculi and recurrent cholecystitis. But it is well known that calculi have developed in a dilated common duct after cholecystectomy (Mayo has observed five such cases); and the danger of fistula persisting after cholecystostomy is not great if no obstruction of the ducts remains.

A further argument in favor of retention of the gall-bladder unless functionally useless is that, should a subsequent operation be required, the gall-bladder is, as stated by Hartmann, not only the thread of Adriadne which guides us through a labyrinth of adhesions to the position of the bile-ducts, but may become a very important feature in the restoration of intestinal drainage of bile, by means of cholecystenterostomy, in cases where the common bile-duct is permanently obstructed. The argument used by the advocates of indiscriminate cholecystectomy in this connection is not valid. They assert that if the gall-bladder had been removed at the first operation no secondary operation for recurrence of symptoms would be required. Such has not been our own experience, and we agree with Richardson that cholecystectomy often is a difficult and dangerous operation—more difficult and dangerous in fact than a simple choledochotomy, were

the latter operation done in cases free from pathological adhesions and in patients not gravely ill with cholemia. The adhesions which form after some cholecystectomies cause more trouble than the original disease.

We believe cholecystectomy is indicated in cases of simple cholelithiasis whenever the gall-bladder presents any gross evidence of disease. When acute calculous cholecystitis (page 503) is present cholecystectomy always is preferable to cholecystostomy; except in cases in which only the slightest operative intervention will be tolerated. When as the result of repeated attacks of acute cholecystitis, or from the long duration of the disease in a latent stage, the gall-bladder is much contracted upon its contained calculi, removal of these and drainage of the gall-bladder even if prolonged seldom will restore to it a sufficient degree of functional activity. In such cases we prefer to do cholecystectomy at once; but we recognize it as a more serious procedure, and if the patient's condition is unfavorable, and in the case of very fat patients, where the operation is one of unusual difficulty, we still practice the simpler operation of cholecystostomy. Should further trouble occur it may be necessary to remove the gall-bladder then; but at all events we hold it is better for the patient to submit to two operations and live to tell the tale than to perish from the first. It is in such cases as these that cholecystectomy is the difficult and dangerous operation to which Richardson refers; but desperate diseases require desperate remedies, and when disabling symptoms persist the gall-bladder must be removed.

Cholecystectomy we see, then, is not indicated in cases of simple cholelithiasis with no gross pathological changes in the gall-bladder: in cases which present evidences of past attacks of acute cholecystitis, and in cases with pericholecystic adhesions removal of the gall-bladder is indicated when operation is done in the interval, unless the constitutional condition of the patient forbids. It is indicated, as we shall point out further on: (1) in most cases of *acute calculous cholecystitis*, as well as in (2) *hydrops with obliteration of the cystic duct*, (3) *chronic empyema*, (4) *calcareous degeneration*, (5) the *cholesterin gall-bladder of* Moynihan and the *strawberry gall-bladder* of MacCarty, (6) *gangrene of the gall-bladder*, (7) *carcinoma*, and (8) in *most cases of perforation*.

Treatment of Biliary Colic.—In many cases the pain is not so severe but that it will be relieved by local application of an ice bag, rest in the recumbent position, and abstinence from all food. Nausea may be relieved by inducing vomiting by drinking a couple of glasses of hot water. If retching persists, lavage of the stomach may be necessary. Should the

pain be great, there is no reason why morphin should not be administered hypodermically; but before this is done the surgeon should be very sure of his diagnosis. If there is a perforation of the stomach or duodenum, instead of an attack of biliary colic, immediate operation will be more effective in allaying pain than many hypodermic injections.

When the attack of biliary colic subsides, the matter of operation should be put before the patient, and he should be urged to submit to having his gall-bladder drained or removed as the surest means of preventing a return of his colic.

Treatment of Stone in the Cystic Duct.—The operation of choice is cholecystectomy. In these cases the stone almost always will be found to have caused ulceration, which will render very probable the subsequent occurrence of stricture. If the gall-bladder and cystic duct are not removed, perforation at the site of impaction in the cystic duct may occur subsequently, and in spite of free drainage of bile from the gall-bladder. Where the duct appears to be permanently occluded, as in cases of hydrops and chronic empyema, then also the gall-bladder and cystic duct should be removed.

Treatment of Stone in the Common Duct.—It has long been considered unwise to operate when there is acute obstruction of the common duct; it is the opinion of the majority of surgeons, in this country at least, that it is better to tide the patient over the attack under medical treatment than to subject him to the danger of an operation when so acutely ill. In Mayo's hands the mortality of operation in such cases was nearly 25 per cent. In all these cases of obstructive jaundice from impaction of stone there are times when some bile filters through and can be detected in the feces by appropriate tests. If the patient has been carried successfully through the stage of complete obstruction, he should be subjected to operation just so soon as the stage of incomplete obstruction is reached. Delay then certainly is more perilous than operation, though even in such cases, where acute cholangeitis persists, but where complete obstruction is not present, the mortality is about 10 per cent.

Treatment during the persistence of acute obstruction is the same as that advised for biliary colic: nothing by mouth; lavage for nausea and vomiting; local applications (ice bag) for pain; morphin for unendurable pain (which is rare); and proctoclysis of normal salt solution or tap water until the stomach becomes retentive and normal persistalsis is restored. Operation during complete obstruction may be forced upon the surgeon sometimes by signs of perforation;

but few such patients will be rescued. Operation should not be done during the existence of spreading peritontis; it should be postponed until the process has become localized.

But though, as we have said, the majority of surgeons still hold to this teaching, we have been forced by our own experience to the conclusion that in the long run *immediate operation during an attack of acute obstruction of the common duct is attended by less danger than is delay.* If operation is delayed the patient runs the risks of cholangeitis, cholemia, with the gravest form of sepsis; not to mention perforation of the common duct or the formation of almost inoperable adhesions; or the indefinite persistence of chronic jaundice with its dangerous hemorrhagic tendencies.

Our experience with immediate operation is so far too limited for us to be willing to erect this as a rule of practice, but while many times there has been cause to regret not operating during the stage of acute obstruction, never yet has there been cause to regret prompt relief of the obstruction by operation.

The operation of choice consists in choledochotomy, removal of the stones (which involves thorough exploration of the common and hepatic as well as of the cystic duct), and drainage of the common duct and of the gall-bladder by separate tubes. Occasionally the gall-bladder has to be removed; but it should be left unless grossly diseased, since should stricture of the choledochus subsequently develop, cholecystenterostomy may be required.

Choledochotomy for the removal of a calculus was first done by Kümmel in 1890. It was studied at length by Terrier in 1892; he distinguished between the operation done for this purpose ("*choledochotomie proprement dite*") and that done for drainage in cases of cholangeitis ("*choledochostomie*"). Parkes in 1885 had proposed the operation. Langenbuch in 1884 had suggested removal of a calculus impacted in the retroduodenal portion of the choledochus by an approach through the transverse mesocolon near the hepatic flexure of the colon. Vautrin, in 1896, reported three cases in which he had employed **mobilization of the duodenum** to facilitate **retroduodenal choledochotomy;** the descending duodenum was loosened from the posterior abdominal wall and turned to the patient's left. The idea of this manœuver appears to have originated with Terrier. Haasler introduced the suggestion into Germany in 1898, and it was finally appropriated and systematized by Kocher in 1903, in connection with his operation of gastroduodenostomy. **Transduodenal choledochotomy** to remove a stone near the ampulla of Vater, proposed in 1884 by Langenbuch, was

first adopted by McBurney in 1891. Kocher in 1894 performed a somewhat similar operation, which he named transduodenal choledochostomy, as he sutured his choledochus incision to the incision in the posterior duodenal wall. Bibliographical references to all these early operations were collected with great care by Terrier in 1892.

Hepaticotomy is the proper term for the operation of opening the hepatic duct; and *hepaticostomy* is often used as an equivalent term with *hepaticus drainage* known also as Kehr's operation (1897), though employed as early as 1889 by Abbe, whose patient was still in good health when the operation was recorded, in 1893.

Hepatotomy, for removal of calculi embedded in the substance of the liver, was employed in 1887 by Knowsley Thornton.

The technique of exposure of the various parts of the common duct and removal of calculi by choledochotomy is described in Chapter XXIV. If possible the calculus should be pushed into the most accessible portion of the common duct, which is then incised directly on the calculus. Sometimes, as urged by Kuhn (1901), it is possible to work the calculus back into the gall-bladder if the cystic duct is dilated, and thus the necessity for opening the common duct may be obviated. When the common duct has been opened it is our custom, and we believe it of much importance, always to make sure that the duct is patent throughout by passing a sound through it into the duodenum, as insisted upon so strongly by Terrier.[1] Until this can be done the surgeon should not be satisfied that he has removed every cause of obstruction. Since adopting this practice we are quite sure that exceedingly few calculi in the common duct have been overlooked.

Drainage in these cases should be prolonged. From four to six weeks at the least should elapse before the biliary fistula is allowed to close; but it often is difficult and sometimes impossible to keep it open so long. It is too early closure of the fistula that is chiefly responsible for recurrence of cholecystitis and cholangeitis after operation.

Treatment of Acute Calculous Cholecystitis.—As intimated already, we have been forced to the conclusion that cholecystectomy is preferable to cholecystostomy in most cases of this nature. Though in cases of cholelithiasis where there never has been an attack of acute cholecystitis, cure without fear of recurrence may be secured in many cases by cholecystostomy, this is not true of the gall-bladder which is acutely inflamed. If prompt operation (within forty-eight hours of the commencement of the attack) is undertaken in cases of acute

[1] Terrier pointed out that this "catheterism" of the biliary passages was first adopted in 1743, by J. L. Petit, in a case of external biliary fistula.

STATISTICS OF CHOLECYSTOSTOMY FOR VARIOUS LESIONS (LANKENAU HOSPITAL)
1912–1920

Lesions of the bile tract	Cases	Died
Abscess, liver	2	0
Calculus, biliary	102	5
Carcinoma, gallbladder	1	0
Carcinoma, gallbladder, chronic pancreatitis	1	0
Cirrhosis liver, cholangitis	1	1
Cholecystitis, acute	1	0
Cholecystitis, acute, calculus	2	0
Cholecystitis, acute, perforated, calculus	1	1
Cholecystitis, acute, calculus, cholangitis	2	0
Cholecystitis, chronic	17	0
Cholecystitis chronic, pancreatitis, acute	1	0
Cholecystitis chronic, pancreatitis chronic	2	0
Cholecystitis chronic, pancreatic lymphangitis		0
Cyst, liver	1	0
Empyema	4	1
Empyema, calculus	5	0
Hydrops, calculus	2	0
Obstruction, common duct, acute	2	2
Pancreatic lymphangitis	3	0
Pancreatic lymphangitis, calculus	12	1
Pancreatitis, acute, calculus	2	1
Pancreatitis acute, cholangitis	3	0
Pancreatitis chronic, calculus	16	0
Pancreatitis chronic, pancreatic lymphangitis	3	0
	208	12 (5.76%)
Cholecystostomy & dochostomy (See dochostomy)	34	3 (8.8%)
Total	242	15 (6.0%)

STATISTICS OF CHOLECYSTECTOMY FOR VARIOUS LESIONS (LANKENAU HOSPITAL)
1912–1920

Lesions of the bile tract		
Calculus	412	26
Calculus, obstruction, common duct, cholangitis	2	2
Carcinoma, gallbladder	2	0
Carcinoma pancreas, calculus	3	3
Cholecystitis, acute	11	1
Cholecystitis, acute, calculus	32	2
Cholecystitis, acute, perforation, calculus	4	2
Cholecystitis, chronic	280	12
Cholecystitis, chronic, cholangitis, hepatitis	1	1
Cholecystitis chronic, carcinoma ileum	1	0
Cholecystitis, chronic, pancreatic lymphangitis	10	1
Cholecystitis, chronic, pancreatitis chronic	5	1
Cholecystitis, chronic, pancreatitis chronic, pancreatic lymphangitis	3	1
Cholecystitis, chronic, pancreatitis chronic, cirrhosis liver	1	0
Cholecystitis, chronic, papilloma, gallbladder	1	0
Empyema	6	1
Empyema, calculus	17	1
Fistula	6	1
Hydrops	5	0
Pancreatic lymphangitis, calculus	40	1
Pancreatitis, acute, calculus	5	1
Pancreatitis, chronic, calculus	9	1
Pancreatitis, chronic, pancreatic lymphangitis	6	0
	862	58 (6.7%)
Cholecystectomy & dochostomy (See dochostomy)	164	11 (6.7%)
Total	1026	69 (6.7%)

STATISTICS OF CHOLEDOCHOSTOMY FOR VARIOUS LESIONS (LANKENAU HOSPITAL)
1912–1920

Lesions of the bile tract	Cases	Died
Calculus	13	4
Cholangitis	1	0
Cirrhosis, liver, acute pancreatitis	1	0
Cirrhosis, liver, calculus	1	0
Pancreatitis, chronic	8	1
Pancreatitis, chronic, calculus	8	1
Obstruction, common duct, stricture	2	0
Obstruction common duct, calculus	1	0
	35	6 (17.1%)
CHOLEDOCHOSTOMY & CHOLECYSTECTOMY		
Calculus	86	6
Calculus, pancreatic lymphangitis	11	0
Calculus, cholangitis, acute	1	0
Calculus, common duct obstruction	2	1
Calculus, common duct obstructln, cholemia	1	0
Cholecystitis, acute	2	0
Cholecystitis, acute, calculus, cholangitis	1	0
Cholecystitis, chronic	12	1
Cholecystitis, chronic, pancreatitis chronic	2	0
Empyema	1	0
Empyema, calculus	1	0
Fistula	2	0
Fistula, empyema	1	0
Fistula, chronic cholecystitis	2	0
Stricture, common duct	1	0
Pancreatic lymphangitis	3	0
Pancreatitis, acute	2	0
Pancreatitis, acute (abscess), calculus	1	1
Pancreatitis, chronic	32	2
	164	11 (6.7%)
CHOLEDOCHOSTOMY & CHOLECYSTOSTOMY		
Calculus	15	1
Cirrhosis liver, cholangitis, calculus	1	0
Cholecystitis, chronic	1	0
Pancreatitis, chronic	1	1
Pancreatitis, chronic, calculus	13	0
Pancreatic lymphangitis, calculus	2	0
Obstruction, common duct, acute	1	1
	34	3 (8.8%)
CHOLEDOCHOSTOMY TOTAL	233	20 (8.6%)

STATISTICS OF CHOLECYSTENTEROSTOMY FOR VARIOUS LESIONS (LANKENAU HOSPITAL)
1912–1920

CHOLECYSTODUODENOSTOMY		
Carcinoma pancreas	5	1
Cyst, pancreas	2	0
Pancreatitis, chronic	4	0
Pancreatitis, chronic, calculus	3	0
Pancreatitis, chronic, cholecystitis, chronic	2	0
Pancreatitis, chronic, cirrhosis liver, duodenal ulcer	1	1
CHOLECYSTODUODENOSTOMY & CHOLEDOCHOSTOMY		
Carcinoma, pancreas	1	0
HEPATICODUODENOSTOMY		
Pancreatitis, chronic[1]	1	0
	19	2 (10.5%)

[1] See Case History, p. 518.

cholecystitis, the gall-bladder and ducts are free from adhesions, and cholecystectomy by the typical technique is an easy operation. Subperitoneal enucleation may be accomplished, after division of the cystic duct and ligation of the cystic artery; and the peritoneal flaps which remain, effectually cover in the bed of the gall-bladder on the under surface of the liver. This complete peritonization of denuded surfaces prevents the development of adhesions, which are almost unavoidable when removal of the gall-bladder is delayed until after the patient has had several attacks of acute cholecystitis. In such cases pericholecystic adhesions already exist, and though the gall-bladder may require removal because no longer functionally useful, the operation is much more difficult and the mortality is much higher than when cholecystectomy is done early in the first attack of cholecystitis. The benign character of the operation under the latter circumstances was first well demonstrated by Leriche and Cotte (1912).

It is in these cases of acute calculous cholecystitis that we find recurrence of symptoms, if not indeed new formation of calculi, after the operation of simple drainage; and we repeat once more that whereas in cases of simple cholelithiasis cholecystostomy is sometimes the operation of choice, in cases of acute calculous cholecystitis cholecystectomy is always to be preferred.

The treatment of the other acute complications of cholelithiasis, such as *perforation*, *gangrene*, *pericholecystic abscess*, etc., is the same as when no calculi are present (page 458).

Obstruction of the Biliary Passages

Obstruction of the biliary passages is of very common occurrence. The causes may be classified in three groups: (1) Obstruction from within the ducts, which is exemplified almost solely by the lodgment of biliary calculi, although rare cases of obstruction from intestinal parasites have been recorded. (2) Obstruction from changes in the walls of the ducts, which are due chiefly to strictures and neoplasms, both rather unusual causes of biliary obstruction. (3) Obstruction from pressure of neighboring structures: among these may be mentioned especially pericholecystic adhesions, pancreatic lymphangeitis and chronic pancreatitis, and carcinoma of the head of the pancreas. Other rarer causes of biliary obstruction must also be classified here—such as pyloric tumors, enlarged lymph-nodes, or kinks of the ducts caused by movable kidney. A large calculus in the cystic duct may by its pressure cause obstruction of the hepatic and common ducts.

Biliary obstruction from impaction of calculi has been considered already: the pathology of the condition was discussed at page 469, and its clinical aspect at page 494.

Strictures of the Bile-ducts.—*Congenital* obstruction of the bile-passages is discussed at page 441. *Acquired* narrowing and occlusion of the bile-passages varies in frequency with the duct under consideration. Stricture of the cystic duct is not uncommon; that of the common duct is rare; while stricture of the hepatic duct is almost unknown.

The **cystic duct** is the least unusual site for a stricture. This is so not only because of its tortuosity, but because it is the commonest site for the lodgment of a calculus; and it is the cicatrization of an ulcer, the result of the passage of a calculus, which is the usual cause of the stricture.

The main *symptoms* presented are those of hydrops of the gall-bladder (page 450), or, if the contents of the gall-bladder become infected, of suppurative cholecystitis or empyema of the gall-bladder (page 451); and *treatment* is that suitable for these conditions.

Stricture of the **common duct** generally is due to injury by the passage or attempted passage of a gall-stone or is the result of a previous operation. It is possible that a stricture may occur as a complication or sequel either of typhoid fever or syphilis.

The *symptoms* of stricture of the common duct are those usually seen in cases of constant obstruction of the duct (page 589), notably jaundice which is constant and increasing, acholic stools, marked indigestion with nausea and emaciation. In the absence of infection above the stricture there should be no fever. The gall-bladder may be distended and palpable.

Strictures of the **hepatic duct** are very rare: theoretically the lesion should cause the same symptoms as those due to constant obstruction of the common duct, with the exception that the gall-bladder will not be dilated. Langenbuch (1897) referred to seven cases found at autopsy. The senior author has met with but one case, occurring in a colleague. The operation consisted in opening the common duct and gradual dilatation of the stricture. The patient remains well, now five years after the operation.

A *differential diagnosis* between stricture of the common duct and other forms of chronic continuous obstruction of the common duct cannot be made with any degree of certainty; nor can a distinction be drawn between obstruction of the common duct and that of the hepatic, except by the absence of a distended gall-bladder in the latter

condition. But as in most cases of cholelithiasis the gall-bladder is contracted, little reliance can be placed on this sign.

The *treatment of strictures of the bile-duct* is by operation, and this should not be delayed, as every day aggravates the patient's condition.

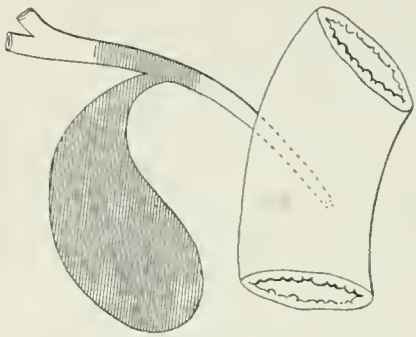


FIG. 143.—Diagram to Show Parts which must be Excised in a Case of Stricture at the Hepatico-cystic Juncture.

Two indications are to be met: to drain temporarily the infected gall-bladder and hepatic ducts, and to reestablish a channel for the excretion of the bile. It was the opinion of Mathieu (1908), to whose excellent monograph on strictures of the biliary ducts every student of this subject must refer, that the former indication can be attained in most cases by the latter means, namely by allowing drainage into the intestine, by direct treatment of the stricture or even by cholecystenterostomy; but should there be any evidence, even the slightest, of active cholangeitis, the surgeon will have to establish independent drainage either by cholecystostomy, or by drainage of the choledochus, or the hepaticus, or even by hepatostomy (page 514).

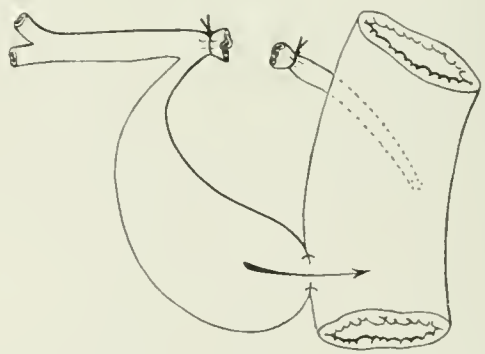


FIG. 144.—Resection of Choledochus, Closure of both Ends and Cholecysto-duodenostomy. (*After Kehr.*)

When there is stricture of the cystic duct, with resulting hydrops or empyema, or an atrophied condition of the gall-bladder, cholecystectomy is the proper operation, combined in many cases with drainage of the common duct. When the obstruction involves the hepati-

cocystic juncture, cholecystectomy must be supplemented by resection of the obstructed portions of the common and hepatic ducts (Fig. 143).

When the stricture is in the common duct or in the hepatic duct, the technique of the operation for the re-establishemnt of the course of the bile comprises: (1) Treatment of the obstruction, when accessible, including resection or incision with plastic operation; and (2) Restoration of the continuity of the outlet, which may comprise end-to-end suture of the divided ends of the duct, or anastomosis of the upper segment of the duct with the intestine and exclusion of the lower segment of the duct.

In cases where the gall-bladder is healthy and the cystic duct patulous, we believe the operation of choice for stricture of the common duct is *cholecystenterostomy* (page 515); thus bile will be discharged into the intestine in spite of the obstruction of the common duct.

Choledochoplasty of the supraduodenal choledochus, analogous to pyloroplasty, was employed successfully by Petersen and by Moynihan, the latter employing temporary drainage of the duct. Stubenrauch (1906) devised a series of plastic operations on the common duct, which are ingenious, but scarcely practicable. In one case he used with success a flap from the stomach to replace the common duct; Kehr employed a similar operation without success, but in another case succeeded in saving his patient's life by employing a flap from the gall-bladder.

Resection of the choledochus seldom has been dome. Oppenheimer (1912) collected eighteen operations, with an immediate mortality of 50 per cent.: ten operations were for carcinoma, with six deaths; four for cicatricial stricture, with two deaths; three for calculus, with no deaths; and there was one fatal operation for benign tumor. Eliot (1917) collected 23 cases of end-to-end suture of the common bile-duct, 4 of which were failures. There were ten primary sutures, the longest defect being 5 cm. (Kehr); and in this case re-operation was required two months later for stricture. There were thirteen instances of end-to-end suture after resection of a stricture, and in these the longest defect bridged was one of 3 cm. (Riggs), the patient in this case being in good health four years and a half later. Although an attempt at end-to-end suture should always be made in these cases, generally it will be sufficient to unite the posterior wall by two or three sutures, and leave a drainage-tube in the opening in the anterior wall. A T-drainage tube is useful in such cases (Fig. 145), and the defect in the choledochus should be covered with omentum. This supports the anastomosis, and the T-tube allows the bile to be discharged both into the duodenum

and through the abdominal wound. When granulations have had time to form, in the course of three to five weeks, the tube is removed through the abdominal wound, and the fistula allowed to close. Much more complicated methods have been used but not with notably better results.

If end-to-end suture of the resected common duct cannot be accomplished, the ends may be joined by a T-tube of rubber, and the gap reinforced by omentum,[1] as already suggested; or Sullivan's method

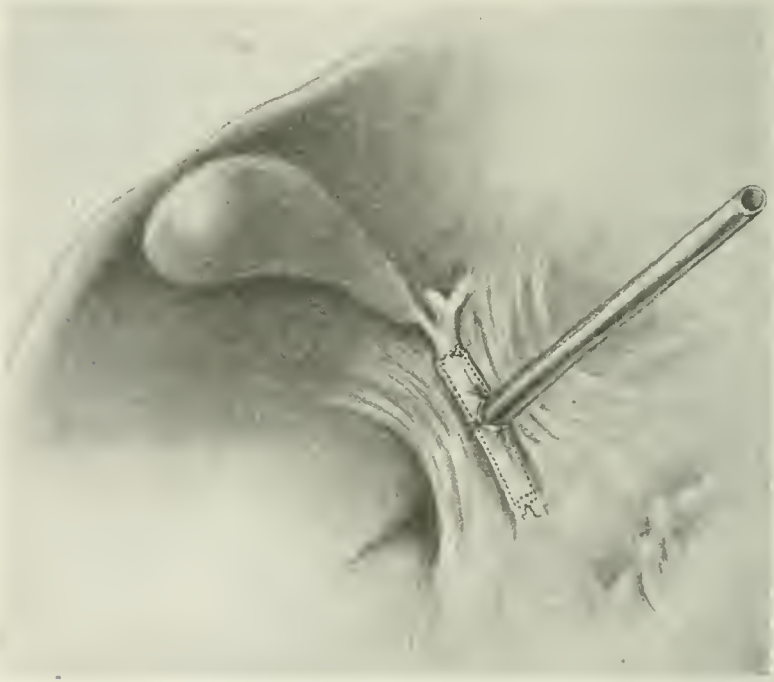


Fig. 145.—After Resection of Choledochus its Posterior Wall has been United by Sutures and a T-drainage Tube Inserted. (*Ginsburg and Speese, Annals of Surgery, 1917, LXV, p. 80, Fig. 2.*)

may be employed (page 516). But if the obstruction is so close to the duodenal termination of the duct that resection cannot be done the duct may be divided above the stricture and its proximal end implanted into the duodenum, the distal end being closed (Fig. 146). This operation (*choledocho-enterostomy*) is less difficult when the duct has become dilated from long-standing back-pressure from a subjacent stricture than in ordinary cases. Termino-lateral implantation, suggested by

[1] Construction of artificial channels for the bile, by means of omentoplasty, was studied experimentally in 1901 by Enderlen and Justi.

Czerny in 1892, is preferable to the original lateral anastomosis employed by Riedel in 1888. If only the hepaticus is available for the anastomosis, *hepatico-enterostomy* may be attempted; it is advisable in such cases to resort to mobilization of the duodenum, and to fix it by sutures to the liver over the site of the anastomosis. Or, as already mentioned (p. 509) a pedicled flap may be turned up from the gall-bladder, the stomach, or from the duodenum (Walton, 1915; Ginsburg and Speese, 1917), and used to construct a channel for the bile from the afferent segment of

FIG. 146.—Choledocho-enterostomy. After Resection of Choledochus Low Down, It may be Possible to Suture Its Proximal End Directly to the Duodenum. (*Ginsberg and Speese, in Annals of Surgery.*)

the bile duct into the gastro-intestinal canal (Fig. 147); or such a channel may be constructed from a free transplant of fascia lata (Fig. 148) or even by means of a rubber tube alone, as indicated at p. 515.

In the first edition of this work we tabulated 42 operations in which an anastomosis was made between the afferent stump of the bile duct and the gastro-intestinal canal: the immediate result is reported in all but one case, there being 30 recoveries and only 11 deaths; but a number of patients who recovered from the operation subsequently developed recurrence of biliary obstruction and all were not relieved by further operation. Guerry (1918) has recently reported seven direct

anastomoses done by himself between the common bile duct and the duodenum, with only one operative death, and four symptomatic cures. The subject has been studied experimentally by Barber (1919).

Some anastomoses have also been made between dilated biliary radicles within the liver substance and the intestine; this operation is known as *hepatocholangioenterostomy* (Fig. 149). It was proposed in 1896 by Baudouin, and in 1897 by Langenbuch and Ulmann. Monprofit in 1904 suggested the propriety of using a jejunal loop in Y. Of

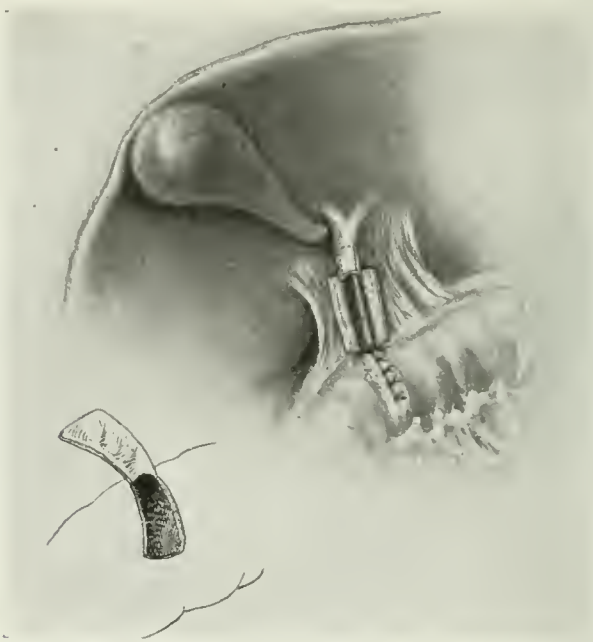


FIG. 147.—Choledochoplasty. A Flap is Turned up from the Duodenum and Sutured to the Proximal End of the Choledochus over a Rubber Tube as a Guide. (*Ginsburg and Speese, in Annals of Surgery.*)

9 such operations tabulated in our first edition, 4 terminated fatally after short intervals, and only one patient is known to have survived more than a few months.

Though *transduodenal choledochotomy* for the removal of gall-stones impacted at the ampulla of Vater has been done on numerous occasions, only a few such operations for stricture are on record (Körte, Oehler); and *excision* of an obstruction at this point seldom has been done except in the case of carcinomata (page 590).

While some form of cholecystenterostomy often is a very successful operation, the same cannot be said of the other procedures mentioned, as will be appreciated by consideration of the statistics already given.

But in view of the extremely poor constitutional condition of the patients, the results are not surprising; and the surgeon certainly is justified in attempting to relieve an otherwise hopeless condition.

Choledochostomy, or union of the dilated common bile-duct with the abdominal wound has been done in a few rare instances, but is no longer a recognized operation. Usually the dilated duct has been mistaken for the gall-bladder itself. The term choledochostomy frequently is used synonymously with drainage of the choledochus. In

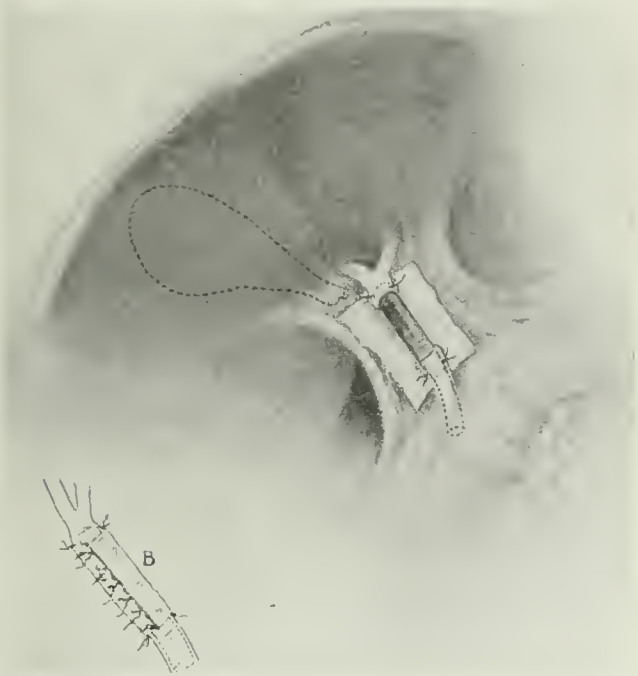


FIG. 148.—Choledochoplasty by Means of a Free Transplant of Fascia Lata. (*Ginsburg and Speese, in Annals of Surgery.*)

the earliest operations on the common duct an attempt always was made to suture the incision made into it for the removal of a calculus; but this was soon abandoned, and *choledochus drainage* is now an operation rather frequently performed in cases of cholangeitis, or after removal of a calculus by choledochotomy. References to the early operations on the common duct (by Kümmell, in 1884; by Thornton and by Heusner, in 1889 and by Courvoisier, in 1890) are given in Berger's monograph. Richter and Buchbinder (1919) urge its suture with two rows of the very finest suture material, and closure of the abdomen without drainage, except in infected cases; and they report

that in only one out of eleven consecutive cases of choledochotomy did they employ a drain. Halsted (1920) also prefers to close the duct without drainage.

Hepaticostomy, or suture of the hepatic duct to the skin for the purpose of drainage has been done a few times under the same circumstances in which choledochostomy was formerly employed. According to Terrier, it was first done in 1889, by Kocher. In Bier's patient death occurred in seven days. In a patient under the care of Mayo the proximal end of the hepatic duct could not be found after an extensive excision for carcinoma; all the bile was discharged by the wound, and death

FIG. 149.—Diagram of Hepato-cholangeio-enterostomy. (*After Kehr.*)

followed at the end of nine weeks. In the somewhat similar case of Kehr, death ensued in seven days from cholemic hemorrhage. The term hepaticostomy sometimes is erroneously used as synonymous with *hepaticus drainage* which is known also as *Kehr's operation* (1897). It is practised quite frequently after choledochotomy, the drainage tube being passed upward past the entrance of the cystic duct, so as to drain the hepatic duct directly (p. 780).

Hepatostomy, or drainage of the intrahepatic bile-passages, is indicated only when all the extrahepatic biliary ducts are strictured or occluded. First adopted by Sendler (1895), in a case of suppurative cholangeitis, it was advocated by W. E. B. Davis (1901) and by Haasler (1904). According to Berger (1903) this operation has been done by Hirschberg, whose technique consisted in performing hepatotomy by a larger trocar, with subsequent dilatation and drainage of

this channel. In some patients with chronic biliary obstruction, the dilated intrahepatic bile-channels project from the surface of the liver in the form of small cysts; and in most of these patients greatly dilated bile-spaces may be found close to the surface of the liver, even if they are not visible on its surface. So the operation is not as erratic as might seem to be the case at first thought. But the operation known as hepatocholangeio-enterostomy (Fig. 149) should be preferred, when practicable. As Mathieu (1908) well says, the operation of hepatostomy is not one of choice, but is comparable to an enterostomy done for intestinal obstruction in the case of a patient too ill to endure the search for the site of occlusion; and, if the patient survives, a secondary operation will be necessary to restore the bile to the intestinal tract.

Cholecystenterostomy.—This operation was introduced in 1882 by von Winiwarter, who made the anastomosis with the colon (cholecysto-colostomy). J. McF. Gaston (1884) and W. E. B. Davis (1901) were among the first in this country to do experimental work in connection with this subject. Until recent times most surgeons have employed the duodenum (cholecysto-duodenostomy); but the operation of cholecysto-gastrostomy was shown experimentally by Oddi (1888) to be well tolerated by dogs after section of the choledochus, and it was adopted in man first by Gersuny (Wickhoff, 1893), then by Terrier (1896) as he found that the presence of the bile in the stomach produced no bad effects, and the anastomosis with the stomach usually was easier than with the duodenum. The jejunum may be used instead of the duodenum, as was done in former years not infrequently; but we believe the advantages of cholecysto-gastrostomy are now quite generally recognized. The statistics of some recent cases of cholecyst-enterostomy in the senior author's service at the Lankenau Hospital have been given at p. 505.

Hepatico-enterostomy by Means of a Rubber Tube.—When the distal end of the choledochus cannot be utilized in re-establishing the normal course of the bile it becomes necessary, when the gall-bladder is absent, to anastomose the proximal stump of the choledochus or the hepaticus with the duodenum. Occasionally this can be accomplished directly by suture (Fig. 146); but often the duodenum, even after it has been mobilized, cannot be brought up near enough to the hepaticus to permit of this method being employed. The use of the jejunum in such circumstances is objectionable, but if an anastomosis can be made with the stomach, this should be done. Several surgeons have had the idea of reconstructing a channel for the bile from

the hepaticus stump to the duodenum or stomach by means of a rubber tube. In some of the earlier operations it was thought necessary to withdraw this tube through the abdominal wound before this was permitted to close. Volleker's technic is indicated in Fig. 150. Sullivan, however, working with Draper Maury, proved by experiments on dogs that it was safe to leave the tube in place, trusting to its expulsion into the duodenum and its final discharge from the rectum.

The plan of operation adopted by Sullivan (1909) may be summarized as follows: The tube used is approximately the size of the com-

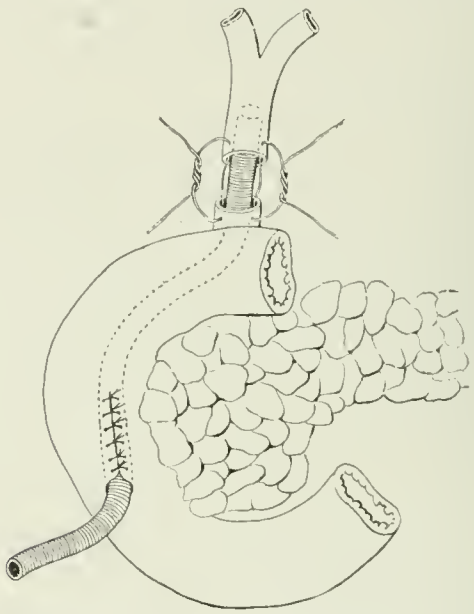


FIG. 150.—Voelcker's Method of Choledocho-(hepatico)-duodenostomy by means of a Rubber Tube which Emerges from the Duodenum through a Witzel Fistula.

mon duct; its duodenal end is tipped with a rubber or marine sponge not larger in diameter than half the lumen of the intestine. The proximal end of the choledochus or hepaticus is freely exposed, and two plain catgut sutures are introduced into one end of the tube, on opposite sides, and are then passed through the wall of the duct from within outward so as to draw the end of the tube into the open end of the duct when the sutures are drawn taut. The tube is carried into the lumen of the duct about one centimetre. The other end of the tube, carrying the sponge, is then implanted into the anterior wall of the duodenum at the level of the papilla of Vater by means of a Witzel fistula (page 319). The tube itself is sutured to the duodenum at one

point with fine catgut so as to prevent its too early expulsion into the intestinal canal. The great omentum is then drawn up and a suitable area is traumatized lightly with gauze friction; similar friction is applied to the duodenum and gastrohepatic omentum on both sides of the tube. The great omentum is then adjusted so as to cover the tube and extend beyond it in all directions, and is held in place by several

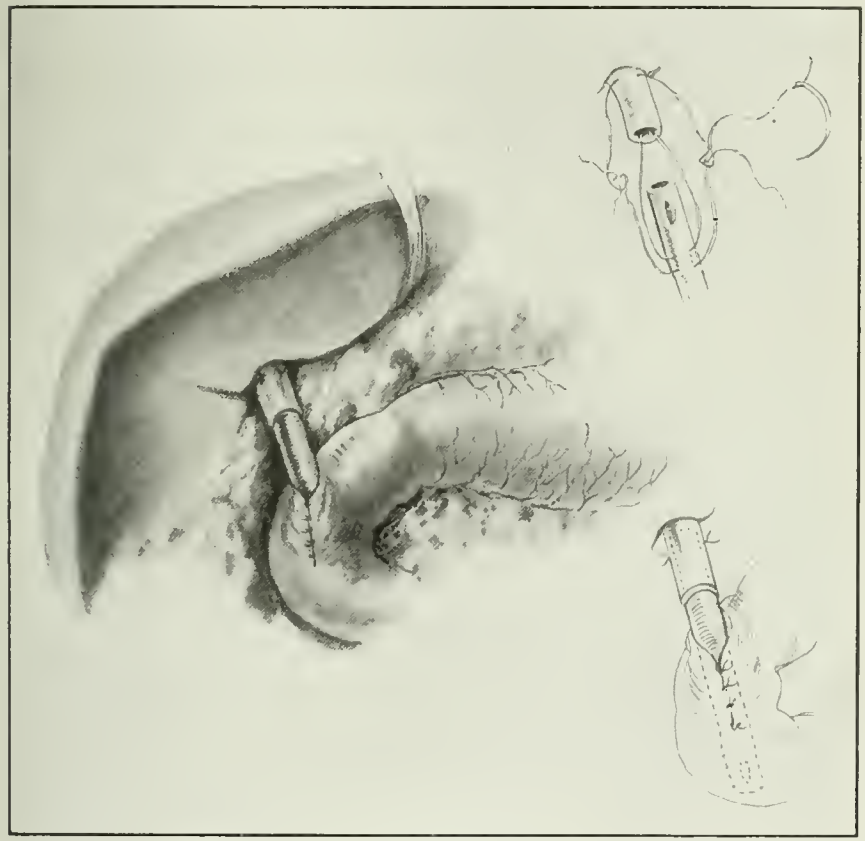


Fig. 151.—Hepatico-enterostomy by Means of a Rubber Tube.

fine catgut sutures. After the retaining sutures of catgut have been absorbed the tube is drawn into the intestine by the tug on the sponge tip exerted by intestinal peristalsis. A more or less permanent channel is formed in this way, permitting the discharge of bile into the intestine. Most surgeons who have adopted this method have not employed any sponge attached to the end of the tube which enters the duodenum, (Fig. 151) and personally we are of the opinion that it is unnecessary.

Case histories reported by various authors, abstracts of which were published in the first edition of this work, show that up to that time (1913) the choledochus had been restored by means of a rubber tube in no less than 12 cases; all the patients survived, but in only 8 does permanent relief appear to have been secured. In several instances the tube never was recovered from the feces, but as it could not be detected by the X-rays it was presumed to have passed unnoticed. Brandt (1912) thinks it desirable that it should remain permanently *in situ*.

The following patients in whom hepatico-enterostomy was required have come under the care of the senior author:

BILIARY CALCULUS, COMMON DUCT OBSTRUCTION, RETROVERTED UTERUS. CHOLECYSTECTOMY; CHOLEDOCHOSTOMY; SUSPENSION OF UTERUS. RECOVERY. COMPLETE OCCLUSION OF COMMON DUCT. HEPATICO-DUODENOSTOMY BY RUBBER TUBE. RECOVERY

C. T., aged 54 years, admitted to German Hospital, January 20, 1914.

Complaint.—Pain in right epigastrum and right costal margin.

Family history.—Negative except for carcinoma (an aunt.)

Social history.—Negative.

Past medical history.—Displacement of uterus several years ago. Menorrhagia. Double nephropexy for floating kidney. Appendicitis five years ago.

Present illness.—Eight months ago patient began to have violent cramp-like pains in the epigastrium and along the right costal margin, radiating to the left shoulder. Pains have at times caused vomiting of sour greenish material. Pain is not related to eating. Has had indigestion and belching of gas for a number of years. Bowels have been constipated since onset of present illness. At present the attacks of pain recur every four or five days and are always accompanied by nausea, vomiting, chills, and fever. Patient has been jaundiced since May last, the jaundice increasing in severity with each attack.

Physical examination.—Abdomen, soft, flat; old operative wound in lower right quadrant. Very tender to the touch in the gall-bladder region.

Operation.—January 21, 1914. Dr. Deaver. Ether anesthesia. Upper right rectus incision. Foramen of Winslow patulous. Head of pancreas slightly enlarged. Gall-bladder small; opened and stones removed. Cystic duct and artery clamped, gall-bladder removed. Probe passed through stump of cystic duct into duodenum. Gall-bladder bed oversewn with iodized gut. Cystic duct and artery tied. One rubber tube with selvage gauze passed into subhepatic fossa. Wound closed in layers to drainage. Lower right rectus incision, etc., suspension of uterus.

Postoperative course.—Patient began to drain bile through tube on second day after operation; drainage continued in large quantities after tube was removed. Stools clay-colored. Patient weak and uncomfortable. Skin still somewhat jaundiced.

February 25.—For past three days patient has been running a septic tempera-

ture. Complains of pain in incision. Purulent discharge from sinus. Drainage still abundant.

Operation.—February 25, Dr. Deaver. Ether anesthesia. Incision through old scar. Viscera packed off with gauze. Strong adhesions to gall-bladder fossa over gastro-hepatic omentum. Search for common duct unsuccessful. Adhesions of omentum to duodenum tied and cut. Duodenum opened and papilla of Vater identified. Common duct had been destroyed, and a probe passed free into peritoneal cavity. One end of a "T"-tube was passed into the hepatic duct and sutured; the other end was passed into the remains of the common duct and sutured with plain catgut suture. Duodenum closed with muco-muscular and musculoserous sutures of chromic gut, reinforced with sero-muscular suture of linen. Bile found to be leaking from proximal end of duct. Probe passed into liver. Great omentum brought over line of suture in duodenum to reinforce duodenum. Duodenum friable and ulcerated from breaking up of adhesions. Rubber tube and cigarette drain passed into subhepatic fossa. Hemostasis with No. 2 iodized gut. Wound closed in layers; skin with silkworm gut. Dry dressing. March 24, 1915. Patient dismissed, with "T"-tube *in situ*, but cut off flush with the abdominal wall. Wound had healed except around the tube.

Blood count (1–20–15).—Hemoglobin, 64 per cent. R. B. C., 4,210,00. W. B. C., 5,700; coagulation time 5 minutes.

(2–24–15) Hem., 74 per cent. R. B. C., 4, 290,000. W. B. C. 11,900; coagulation time 8 minutes.

Culture of bile.—B. coli com.

Culture of duodenum sterile.

The patient returned to the hospital November 11, 1915, still wearing the "T"-tube. For about six weeks after operation the jaundice had cleared but soon reappeared. At present she has periodic attacks of chills and fever, with deepening jaundice, sometimes clearing partially and again growing deeper. Patient complains of severe diarrhea and intense itching which, to use her expression, sets her crazy; distension, flatulence, belching, and frequent vomiting. Describes feeling in right hypochondrium as though "something were giving away." Appetite fair, bowels constipated, stools light, urine dark. Has not been able to work since operation.

Physical examination.—No masses palpable, but there is a feeling of resistance and rigidity in the upper right quadrant. Peristalsis normal. Liver and kidney not palpable. Jaundice deep.

Blood pressure, 130–78.

Blood count.—(11–19–15). Hem. 60 per cent. R. B. C. 3,730,000; W. B. C. 7,100; coagulation time 7 minutes.

Operation.—November 22, 1915. Dr. Deaver. Ether anesthesia. Curved incision around old scar. Old scar removed. Old "T"-tube left in tract to serve as a guide. Peritoneum opened. Peritoneum adherent to stomach, and duodenum adherent to liver. The great omentum had to be delivered to get the relations. The old "T"-tube communicated with the liver but not with the common duct. The hepatic duct was located with much difficulty; opened and great quantities of fluid, such as comes from a hydrops of the gall-bladder, exuded. A rubber tube, about the calibre of the little finger, was sewn into the hepatic duct, and the other end of the tube was sewn into the opening in the duodenum which the old "T"-tube

had made, an anastomosis between the hepatic duct and the duodenum being thus formed. One rubber tube was put into the renal well; another down to site of anastomosis. Wound closed in layers to drainage with No. 2 iodized gut; skin with silkworm gut. Dry dressing.

Patient's jaundice cleared entirely immediately after operation. Recovery was uneventful.

The patient was seen again six months after her dismissal from the hospital. She was gaining in weight and was in excellent health.

Chronic Cholecystitis; Internal Biliary Fistula. Repair of Duodenum. Cholecystectomy. Gastro-jejunostomy. Recovery. Recurrence of Obstructive Jaundice; not Relieved by Hepatico-duodenostomy

I. W., female, aged fifty-five years, admitted to German Hospital, June 7, 1914.

Past medical history.—Jaundice 10 years ago but does not remember any attacks of pain. Pneumonia 10 years ago. Typhoid fever 5 years ago.

During the past ten years she has had pain, not sharp, in the right upper abdomen, with a constant dull heavy feeling. No chills, fever, or jaundice. Becomes nauseated after eating but does not vomit. Is constantly distended with gas, and belches considerably. Appetite poor. Bowels very constipated. No loss of weight. Dry cough since March, 1914.

Physical examination.—Tenderness and moderate rigidity over ninth right costal margin, and some tenderness in epigastrium.

Operation, by Dr. Deaver, June 7, 1914. Ether anesthesia. Upper right rectus incision. Gall-bladder adherent to duodenum and a direct fistula was present. The gall-bladder was separated and the opening in the duodenum invaginated with linen. A thickened and contracted gall-bladder removed. Bed over-sewn with number 2 iodin gut. Adhesions at duodeno-jejunal junction freed. The duodenum having become contracted by invagination of the fistula, a posterior gastro-jejunostomy was done. One glass tube in subhepatic fossa. Wound closed in layers. Dry dressing. Patient left the hospital, July 7, 1914, against advice. The wound was granulating at the time.

She was re-admitted to the German Hospital, September 9, 1914. Two weeks after leaving the hospital she had an attack of severe cramp-like pain all over the abdomen, accompanied by fever, chills, vomiting, jaundice. She has not felt well since that time and has had several subsequent attacks of chills, fever, and jaundice, with pain in the right hypochondrium radiating to the back. Eating is regularly followed by pain in the epigastrium and by nausea and occasional vomiting, which sometimes relieves the condition. Belches considerable gas. Jaundice is now constant; stools are clay colored, urine dark; and skin itches. Has lost weight.

Physical examination.—Shows the scar of the previous operation also tenderness and rigidity in the epigastrium and the right hypochondrium.

Operation.—September 15, 1915. Dr. Deaver. Ether Anesthesia. Incision around old scar. Adhesions of omentum to liver and intestines cut and tied. Cystic degeneration of omentum. Gastro-jejunostomy opening in good condition. Pancreas normal. No stones found in common duct. Common duct aspirated and incised, a cloudy watery fluid escaping. Culture taken. Hepatic duct probed

and patulous. No opening found in distal end of common duct. Duodenum incised and "T"-tube placed from hepatic duct to duodenum. One cigarrette drain placed below omentum, and omentum brought down over duodenum. One piece of rubber-dam to subhepatic fossa. Wound closed to drainage. Skin with silkworm gut. Dry dressing. Patient transfused, 1800 cc. saline, on table.

External biliary fistula had not completely closed when the patient was discharged, October 22.

The patient returned to the hospital during the winter, complaining of the same symptoms as before and wanting to undergo another operation, which Dr. Deaver did not deem advisable. Death occurred about six months after the hepatico-duodenostomy, jaundice having persisted until the end.

Biliary Obstruction from Causes Outside of the Ducts.—The most frequent of these causes is the presence of pericholecystic adhesions (page 452), which cause kinking of the cystic or common duct. The symptoms and treatment of this condition have been considered at pages 452 and 483.

Obstruction from diseases of the pancreas is discussed at page 694.

Obstruction from carcinoma of the papilla of Vater is not very rare (page 588).

Obstruction from kinking of the ducts due to the presence of a *movable kidney* was first recognized by Wiessker, in 1888, and was studied by Tinker (1907). According to Tinker, Apolant believes that many of the patients treated at Carlsbad for supposed affections of the biliary tract are in reality suffering from pressure of a misplaced kidney. Tinker reports two cases of his own in which the patients secured immediate and permanent relief of all symptoms referable to the biliary tract after the operation of nephropexy. The *symptoms* presented are those of interference with the normal flow of bile, indigestion, nausea and occasionally vomiting, pain in the right hypochondrium referred to the back or shoulder, and in some cases intermittent jaundice. Although the symptoms all point to the biliary tract, they are relieved by the return of the kidney to its normal position, either by manipulation, or when the patient assumes the recumbent position.

The *diagnosis* usually is not made until operation has demonstrated that there are no lesions in the biliary tract. The possibility of a movable kidney as a cause should be borne in mind, and in cases of doubt this factor should be eliminated by rest in bed or the use of a well-fitting corset with a kidney pad, before operation is undertaken. When it has been ascertained definitely that a movable kidney is the cause of the biliary symptoms, the operation of nephropexy should be advised.

Obstruction from fibro-adenoma forming in the stump of the cystic

duct after cholecystectomy was encountered by Mayo (1915) in two cases.

Obstruction from Aneurysm.—This is a rare cause of biliary obstruction. According to Villandre (1909), most cases have developed shortly after some acute infection, especially pneumonia or typhoid fever. The aneurism may effect the abdominal aorta, or the heptaic artery or one of its branches. Fleckenstein (1913) says Bosdorf (1889) found one aneurysm of the hepatic artery among 93 aneurysms encountered at autopsy on 3108 adults; and that Miller (1902) found 3 among 171 aneurysms in 6425 autopsies on adults. Verrey (1911) collected 42 cases of aneurysm of the hepatic artery, and Fleckenstein's case made the forty-third on record, but there is a reference at least to one case in nearly every recent volume of the Index Medicus. The symptoms to attract attention usually have been those of pressure, and not any symptoms attributable to the aneurysm as such. In most cases the diagnosis has been made only at autopsy. Villandre thinks that if the triad of symptoms (local pain, intestinal hemorrhages, and jaundice) is present, the existence of an aneurysm of the hepatic artery should be suspected. These are the most characteristic symptoms, but one or more often is absent. Intestinal hemorrhage alone may be attributed to duodenal ulcer, and even when associated with local pain and jaundice might be due to malignant obstruction of the common duct.

Villandre collected the following cases of aneurysm of the hepatic artery in which operation was done:

Mikulicz: lesion not found; gastro-enterostomy. Death in six days. Diagnosis at autopsy.

Riedel: incised aneurysm, thinking it was calculus in common duct; tampon for hemorrhage. Second operation, twenty days later, abandoned and tamponed for hemorrhage. Death in three days. Diagnosis at autopsy.

Heller: found blood in abdominal cavity, mesocolon, and in gall-bladder and cystic duct. Tamponed. Death.

Habs (report by Grunert): after cholecystectomy recognized aneurysm of hepatic artery as cause of choledochus obstruction. Nothing further done. Death in eight days.

Allessandri: cholecystotomy; hemorrhage; tampon. Death in five days.

Tuffier: incised mass in gastro-hepatic omentum; profuse hemorrhage; ligated hepatic artery. Death in four days.

Kehr: incised gall-bladder; gush of blood, controlled only by ligation of hepatic artery. The gall-bladder was then extirpated and sac of aneurysm packed. Aneurysm had ruptured into gall-bladder and further hemorrhage prevented only by plugging of cystic duct by clot. Recovered, and according to Bode in good health six years later (Fig. 152).

To these may be added an operation by Garré, reported by Bode: (1909) over a year after an injury to the right thorax Garré operated on a patient who presented symptoms of pyloric obstruction with gastro-intestinal hemorrhages. The diagnosis was gastric ulcer, and gastro-enterostomy was done. The patient died a week later, and at autopsy an aneurysm was found on an intrahepatic branch of the hepatic artery; this had ruptured into one of the intrahepatic bile-ducts, but there had been no recent hemorrhages. There were scars of old injury in the liver and right kidney.

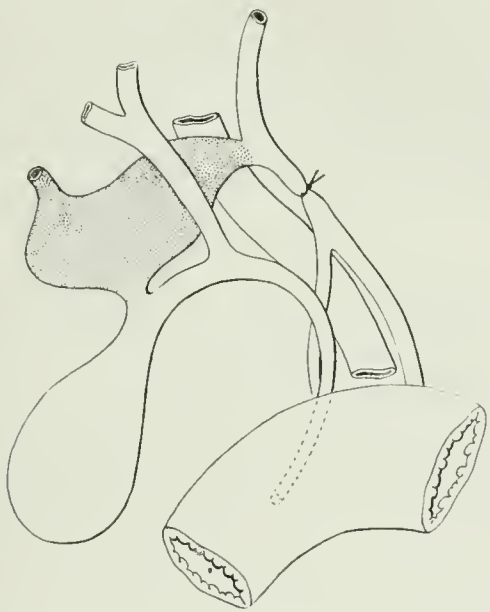


FIG. 152.—Kehr's Case of Aneurysm of the Hepatic Artery which had Ruptured into the Gall-bladder. Ligature on the Hepatic Artery.

Baruch (1914) also reported a case, in which, at operation undertaken for symptoms of obstructive jaundice without palpable enlargement of the gall-bladder, a compressible tumor, the size of a goose-egg, was found and was mistaken for the choledochus; puncture, however, drew pure blood. After cholecystectomy it was impossible to insert a probe into the cystic duct, so after mobilization of the duodenum the hepatic duct was exposed. It was drained above the point of obstruction by the aneurysm, but the patient died the next day. Autopsy showed the aneurysm involved the hepatic artery on the liver side of the origin of the gastro-duodenal artery.

Rational operation (ligation of the hepatic artery) was done only in two cases, those of Tuffier and Kehr.

Kehr's is the only patient who recovered. Villandre conducted experiments on dogs, to ascertain the feasibility of survival after ligation of the hepatic artery. Though few in number these experiments tended to demonstrate that while gradual occlusion is safe, yet rapid occlusion, such as occurs in embolus or after ligation of the normal

artery, always is fatal. Evidently in Kehr's case adequate collateral circulation had been established before the operation, only a segment of the liver along its anterior border becoming necrotic. It is noteworthy that Kehr has also successfully ligated the hepatic artery in one case for hemorrhage during choledochotomy.

Körte did cholecystenterostomy for obstruction from a mass at the papilla of Vater (probably a congenital stricture); death occurred from internal hemorrhage, and at autopsy a thrombosed aneurysm was found in the course of the bile-ducts. This had not caused any symptoms.

Portal thrombosis, according to Bode (1909), has been treated by operative means in eight cases, only three patients recovering—two after the Talma operation for ascites from cirrhosis of the liver (epiplopexy), and one after simple drainage of the ascitic fluid.

Biliary Fistula

A **biliary fistula** is one that discharges, or has discharged bile. Such fistulæ are not uncommon. They result in most cases at the present day from operations on the biliary tract; but they may also be caused by inflammatory changes with perforation, or from carcinoma of the gall-bladder or bile-ducts. They are classified as *internal* or *external*. External fistulæ are those which open upon the surface of the body; internal fistulæ are those which communicate with one of the internal organs.

Courvoisier (1890) collected the following statistics in regard to the location of biliary fistulæ in 499 cases.

Courvoisier's Statistics of Biliary Fistula

External fistulæ	196 cases	
Internal fistulæ	303 cases	
Between biliary tract and peritoneal cavity	70	127
Between biliary tract and peritoneal adhesions	49	
Between biliary tract and retroperitoneal tissues	3	
Between biliary tract and portal vein	5	
Between biliary tract and thoracic organs	24	24
Between biliary tract and urinary organs	7	7
Between biliary tract and other abdominal viscera:		
Stomach	13	137
Duodenum	83	
Jejunum	1	
Ileum	1	
Colon	39	
Between biliary tract and other portions of biliary tract	8	8
Total internal fistulæ		303

Internal Biliary Fistula.—A fistulous opening between the biliary tract and one of the adjacent viscera may occur after an adhesive peritonitis has bound the two structures together; this forms the *direct* variety of internal fistula. If the communication occurs through an abscess cavity, the affected viscera not being in direct contact, the fistula is said to be of the *indirect* variety. As a rule, perforations of the biliary tract into the free peritoneal cavity or into peritoneal adhesions are not classed as biliary fistulæ, though they are included in Courvoisier's statistics quoted above.

A true fistula may exist between two or more of the biliary passages themselves; between the biliary passages and the substance of the liver or pancreas; or the fistula may join the gall-bladder or ducts with the lumen of the stomach, duodenum, colon, or some portion of the small intestine. Robson (1909) observed during a period of six years no fewer than five cases of internal biliary fistula: 3, between gall-bladder and duodenum, and 2 between gall-bladder and stomach. In one of the latter cases the gall-bladder had prolapsed into the stomach through the fistula. There are on record a number of cases of biliary fistulæ communicating with the urinary, thoracic (pulmonary, pleural, pericardial, mediastinal), and female pelvic organs, but these are pathological curiosities and have comparatively little interest for the surgeon. Usually they have been found at autopsy, or have developed so soon before death that no attempt at operative relief has been justifiable. They are much rarer now than in the period before diseases of the biliary tract came under the domain of surgery.

Symptoms.—When the biliary fistula is of such size as to permit the discharge from the gall-bladder of all its contained calculi, with free drainage of the infected biliary tract, all inflammatory symptoms quickly subside. Nature's cholecystenterostomy often is as successful as an operative anastomosis in relieving the patient of distressing symptoms. The true condition of affairs may be surmised, if a patient passes by rectum one or more calculi too large to have traversed the bile-ducts; in one case on record the diagnosis was made from the fact that the patient vomited gall-stones. In this case recovery ensued without operation.

The following cases represent progressive stages in the development of internal biliary fistulæ:

PERICHOLECYSTITIS; CALCULI AMONG ADHESIONS AROUND GALL-BLADDER, CHOLECYSTOSTOMY. RECOVERY

R. Z., female, aged thirty years; admitted to the German Hospital November 22, 1906. Mother died of carcinoma of the liver. Patient had typhoid fever when

twenty years old. Bowels always constipated. For fifteen years has had attacks of pain in epigastric and gall-bladder regions, with vomiting. No jaundice until four months ago, when she had jaundice with chills and fever.

Examination.—No jaundice on admission. Liver normal; gall-bladder palpable. Tenderness and rigidity in gall-bladder area. W. B. C., 10,000.

Operation by Dr. Deaver. Ether anesthesia. Incision splitting fibres of upper right rectus. Adhesions around gall-bladder, and between it and the omentum. On the outside of the wall of the gall-bladder were several gall-stones which seemed to have worked their way gradually through the walls of this viscus. They seemed to be very lightly attached to the gall-bladder and fell off during light manipulation. The gall-bladder was distended and packed with stones. Stones removed and gall-bladder drained with rubber tube. Recovery. (Note: Plate VI shows a similar condition in a patient treated by cholecystectomy.)

Cholelithiasis; Cholecysto-gastric Fistula. Cholecystectomy; Repair of Stomach; Drainage of Choledochus. Recovery

Mrs. W. D., aged 42 years; admitted to the German Hospital April 10, 1911. Has had numerous attacks of biliary colic, extending over many years. Has made several trips to Carlsbad, always with temporary improvement. Operation undertaken because of persistent recurrence of attacks.

Operation by Dr. Deaver. Ether anethesia, preceded by nitrous oxide. Incision splitting fibres of upper right rectus, extended inward along costal margin. Duodenum and pylorus bound by strong adhesions to fundus of gall-bladder and lower surface of liver. On releasing adhesions a perforation of stomach and of gall-bladder was found, with a large gall-stone protruding into stomach. Adhesions freed and opening in stomach closed. The free border of gastro-hepatic omentum was next opened, exposing cystic and common ducts. Cystic duct and artery ligated and cut, and gall-bladder removed, after calculus had been pushed back into gall-bladder. Gall-bladder was thickened and contracted. A large stone, the size of end of thumb, occupied gall-bladder cavity. Mucosa of gall-bladder edematous and ulcerated. Margin of cystic duct grasped with hemostat and probe passed down common duct to duodenum; hepatic ducts also free from obstruction. Small rubber tube sutured into cystic duct opening with chromic catgut. Gastro-hepatic omentum then closed around tube. Split tube and piece of gauze then sutured into gall-bladder fossa on under surface of liver, sutures passing through liver substance. Small glass tube to sub-hepatic fossa. Uneventful recovery.

Most of the internal fistulæ tend to close spontaneously, but the resulting adhesions often cause very distressing symptoms. These adhesions may cause pyloric obstruction, kinking of the common duct, interference with intestinal peristalsis, or even acute intestinal obstruction. If the internal biliary fistula remains patulous, and if the cystic duct is patulous, obstruction of the common duct may not produce any noteworthy symptoms, as the bile will be able to drain into the intestinal tract through the fistula. When the bile is discharged into the colon there are more digestive symptoms than when it

escapes into the duodenum or stomach. Interference with the function of the pancreas may occur, and may eventually demand operative relief. In a case reported by Leonardi (1909) the entire gall-bladder was discharged as a slough into the intestine through an internal biliary fistula, and was passed by rectum.

Treatment.—As the true lesion scarcely ever is recognized until after the abdomen has been opened, the surgeon must be prepared to meet the conditions as they exist. It is seldom possible and very rarely is it proper to return the various viscera involved in the adhesions to their normal relations. In most cases the intestinal or gastric opening can be closed by careful suture; if this narrows the lumen to a dangerous degree a short circuiting operation should supplement the repair of the fistulous opening. Only in rare cases is an intestinal resection required. The biliary side of the fistula is most easily relieved by cholecystectomy, when the fistula communicates with the gall-bladder or cystic duct; in such cases as well as in those where the common or hepatic duct are involved in the fistula, drainage of the main bile-channels (choledochus or hepaticus) should be instituted.

External Biliary Fistulæ.—Most of the external biliary fistulæ encountered at the present day are the result of an operation, and develop in the operative cicatrix. Those which form spontaneously, and which sometimes are called "pathological," may open almost at any point of the abdominal wall. The situation of the skin opening depends upon the size and position of the gall-bladder, and upon whether or not the fistula communicates directly with the gall-bladder or ducts, or passes through an intervening abscess cavity by a long and tortuous channel. In the series of cases collected by Courvoisier (1890) the fistulous openings were in the following situations:

In the right hypochondrium	49
At right costal border	36
On right side of epigastrium	17
In epigastrium	6
In right iliac fossa	10
Near umbilicus	22
At umbilicus	11
In left groin	1
Multiple openings	1
	153

External biliary fistulæ are classed as *complete* (*biliary*) or *incomplete* (*mucous*), according to whether or not they discharge bile. Practically all the spontaneous or so-called pathological fistulæ are complete.

The diagnosis is made by the recognition of bile in the discharge. In most cases calculi also escape from time to time. In a case reported by Gutteridge (1878) a single stone 3 inches in diameter was discharged in this way. Usually such a fistula persists until all calculi present in the biliary passages have been discharged; it may then close of itself. But the period of time during which it will remain open cannot be foretold, and in most cases early operation is indicated to remove the remaining calculi, and restore the intestinal drainage of bile.

Abscess of Abdominal Wall from Perforation of Gall-bladder; Calculi in Gall-bladder. Cholecystostomy. Recovery

M. R., female, aged forty-one years; admitted to German Hospital, October 20, 1904. Two brothers had had gall-stones. Patient never had had typhoid fever. Present illness began twenty-three years ago, with severe pains in epigastrium and vomiting; indigestion after eating, accompanied by much flatulence. Last attack of severe pain five years before present one, which has been more or less constant during last five months with dull pain in gall-bladder region referred to back; lately pains have been sharp and shooting.

Examination.—Edema of abdominal wall in gall-bladder region, with swelling, redness and tenderness. Marked tenderness in right hypochondrium. Hemoglobin, 61 per cent., W. B. C., 9200.

Operation, by Dr. Deaver. Ether anesthesia. Incision over mass in abdominal wall which proved to be an abscess communicating with fistulous opening in the gall-bladder. Adhesions between gall-bladder, stomach, duodenum, and colon. Adhesions not disturbed. Liver enlarged and congested. Three small and one large calculi removed from gall-bladder through fistula. Drainage of gall-bladder. Recovery.

Postoperative fistulæ may be either mucous or biliary.

A postoperative mucous fistula is very rare if the gall-bladder has been removed; usually it indicates that the cystic duct is no longer patent, and that the gall-bladder is a useless appendage. The cystic duct usually is closed by cicatricial changes which are the result of previous disease, but in some instances closure is due to impaction of a calculus. There is a more or less constant flow of muco-purulent material from the fistula, but the discomfort produced may be very slight, the amount of the discharge seldom being more than 30 to 40 c.c. (1 ounce) in twenty-four hours. Should the external opening close, however, the discharge will accumulate within the fistulous tract, causing severe pain and at times symptoms of septic absorption. Should operation be inadvisable for any reason, or should it be refused, the external opening of the fistula should be kept patulous by the use of a tube. The following case illustrates this condition, as well as the method or cure by operation.

Mary S., aged twenty-seven years, was operated upon in the German Hospital in 1908, for gall-stones, a cholecystostomy being performed. The sinus was almost closed when she went home. Shortly after it closed she had severe crampy pains in the region of operation. The fistula was opened, with immediate relief, and a rubber drainage tube was inserted into the tract. This was worn by the patient for over two years. The discharge consisted of white muco-purulent material. Two weeks before readmission to the German Hospital, the patient noticed a discharge of bile which continued profusely until the second operation.

Second operation, April 28, 1911. Ether anesthesia. Incision made around old cicatrix, and the fistulous tract dissected down to the gall-bladder. The gall-bladder was removed, and all the ducts explored, with negative result. All the ducts were patent, and no calculi were present. Patient went home June 4, 1911, with the wound entirely healed.

In the *postoperative biliary fistula* a more serious condition is present. Usually there is an obstruction in the common duct, and bile is discharged continuously from the external opening of the fistula; as much as 1000 c.c. (one quart) of bile may be lost in twenty-four hours. The deleterious effects of complete loss of bile from the intestinal tract were studied experimentally in dogs by Pawlow in 1905; he found that health was rapidly lost and bone atrophy occurred. Seidel (1910) reported two similar cases in man. But if the discharge is not very profuse, the patient may be in very good health, although in most cases there is a certain amount of indigestion, and the patient complains of feeling "miserable." The discomfort caused by the discharge of bile usually is so great that operative measures should be instituted for its relief. But in a certain number of cases external drainage of bile after operation acts as a therapeutic measure in relieving the angeio-cholitis found at operation, and by permitting a subsidence of pancreatic lymphangeitis. In such cases it may be necessary to wati six to eight months or a year before it will be entirely safe to restore the bile to the intestinal tract; and before this time has elapsed the fistula may close spontaneously. So long as bacteriological examination shows the biliary discharge to be actively infected, we believe it is inadvisable to undertake operation for its relief. Before operation is adopted even in cases where the discharge proves sterile, it is well to try irrigation of the tract. In this way it often is possible to dislodge calculi which may have been overlooked at the time of operation, or which may have descended from the intrahepatic bile-ducts since that time. The fluid used for irrigations may be olive oil, or a 0.5 per cent. solution of animal soap, as advised by Brockbank; or a solution of turpentine in ether, as recommended by Robson, especially when the obstruction is due to the presence of stones or fragments. A small soft catheter is passed

down the fistula until an obstruction is encountered, when the irrigating fluid is forced gently into the catheter by the force of gravity, or from a syringe.

When operation is undertaken, the fistulous tract shoud be dissected out cautiously until the peritoneum is opened. Surrounding structures are then packed off, and the dissection continued until the biliary system is exposed. If an inoperable obstruction of the common duct is found, the surgeon will be very fortunate if a reasonably healthy gall-bladder has been left at the previous operation, since it may now be used to conduct the bile into the intestinal tract by means of a cholecystenterostomy. If the gall-bladder has already been removed, or if it requires removal at the second operation, some form of anastomosis will now have to be made between the common or hepatic duct above the obstruction, and the intestinal tract (page 515). If the obstruction of the common duct can be removed, this should be of course done; a calculus may be extracted or pushed into the duodenum; a stricture may be stretched or incised; a benign or even a malignant tumor may be extirpated. Where restoration of the discharge of bile through the common duct can be secured, the gall-bladder should be removed. These operations never are easy, and may be very difficult.

Biliary Fistula. Cholecystectomy; Choledochotomy. Death

B. S., female, aged thirty-eight years, admitted to the German Hospital September 16, 1912, with a history of having been operated upon for gall-stones nine months previously. A biliary fistula persisted. There had been no attack of pain or jaundice. Bowels had been regular, but the stools had been light in color, soft and of foul odor. The fistula discharged dark brown fluid mixed with mucus.

Operation, September 18, 1912. Ether anesthesia. Scar dissected out. Adhesions found between the omentum, gall-bladder, under surface of the liver, and the abdominal wall. The liver was adherent to the parietal peritoneum and could not be displaced. Adhesions ligated and divided, and gall-bladder exposed. It was of good size, but its walls were markedly thickened and a stone was encysted in its wall. Cholecystectomy. Large stone found in common duct. Choledochotomy, stone removed, and duct drained. Owing to the fixity of the liver it was impossible to ligate the cystic artery because of its depth, and two hemostats were left in place. Drain placed in bed of gall-bladder and in the subhepatic space, and the wound in the abdominal wall partly closed. The patient did not react from the operation, and died twenty-four hours later.

Intestinal Obstruction from Gall-stones

Intestinal obstruction due to gall-stones is a complication or sequel of cholelithiasis that is not frequently seen, although it is not extremely rare. Statistics from various sources show that gall-stones are the

cause of from 2 to 4 per cent. of cases of intestinal obstruction. In a large majority of cases the calculus enters the intestinal tract through a fistulous opening between the gall-bladder and duodenum.

Porter (1906) suggested the following as a convenient classification of cases of intestinal obstruction from gall-stones: *Directly*, by plugging or corking the bowel (obturation): *indirectly*, (1) by causing intestinal paresis; (2) by the production of volvulus; (3) by producing a stricture of the intestine; (4) by causing spasmodic contraction of the circular muscle fibers of the bowel; and (5) by producing angulation of the bowel.

The most frequent cause is *obturation*, or the impaction of the calculus in the intestine; the next most frequent causes are *volvulus* caused by violent peristaltic movements of the intestine in the effort to rid its lumen of the obstructing body; and *strictures* the result of ulceration caused by gall-stones which have been arrested for a time, but which may have passed by rectum long before the stricture gives rise to symptoms. Occasionally gall-stones in their course through the intestinal tract may become lodged in *diverticula*. Dr. Henry Winsor has shown us photographs of the specimens from a remarkable case which came under his observation at autopsy, while in Manila, P. I. The small intestine presented innumerable diverticula in which were lodged concretions presumably biliary in origin.

Obstruction from obturation by gall-stones may occur in any portion of the intestinal tract from pylorus to anus. As the small intestine gradually narrows from its beginning to the ileo-cecal valve, the position of the obstruction naturally varies with the size of the stone. Large calculi are arrested higher than smaller, and the latter may succeed in escaping from the body through the anus after causing slight or partial obstruction at various times. Very small calculi may be passed without producing symptoms of obstruction at any time. A very large calculus, measuring $4\frac{1}{2}$ by $3\frac{1}{2}$ inches in circumference was found impacted in the ileum in a case recorded by Roberts (1903), and Baildon (1915) removed one measuring $5\frac{1}{2}$ inches in circumference.

According to Courvoisier's statistics (1890) of fifty-two cases, the site of impaction was in the duodenum and jejunum in 21.4 per cent.; in the ileum in 65.4 per cent.; at the ileocecal valve in 10 per cent.; and in the sigmoid flexure in 2.4 per cent. LeBec and Müller (1903) recorded a case of pyloric obstruction from impaction of a gall-stone.

The **symptoms** are those of intermittent obstruction, the obstructing body being more or less migratory. When obturation occurs, obstructive symptoms are noted at once. The initial symptoms may be very mild. Slight colicky pains, quickly subsiding, and leaving the patient

apparently in perfect health, may occur for many hours before anything more serious is noted. Nausea appears early, and is followed by vomiting. Distention of the abdomen, which never is an early symptom, may never occur at all if the obstruction is very high in the intestinal tract. When the proximal portion of the bowel becomes very much distended, or reversed peristalsis is present, the calculus may slip backward and relieve the obstruction. Under such circumstances the symptoms gradually subside and the patient returns to an apparently normal condition; but will again become a victim of obstruction when the concretion is forced down into a portion of the bowel with lumen too small to accommodate it.

In some cases the calculus can be felt through the abdominal walls as a round or slightly oblong hard mass, freely movable, and devoid of tenderness. The position of this mass may be in any portion of the abdomen or in the pelvis, in which latter position it may readily be mistaken for an ovarian neoplasm.

The **diagnosis** of gall-stone obstruction of the intestines cannot be made with certainty in most cases until the abdomen has been opened. A history of cholelithiasis, if obtainable, a history of previous similar attacks of obstruction, and the presence of a more or less movable hard mass, may lead to a recognition of the true condition.

The **treatment** is operative, except in a few cases where the symptions of obstruction have been very mild and not of long duration. In such cases it may be advisable to temporize, trusting to Nature to expel the calculus. Under no circumstances should purges be given. If operation is not required, it is best to take measures to check peristalsis by administering nothing whatever by mouth, by applying ice to the abdomen, and even by giving morphin hypodermically in the hope that, intestinal spasm being relieved, the stone may pass onward without causing further trouble. If symptoms of obstruction are at all severe, however, operative treatment should be undertaken at once. Unless the obstruction can be accurately localized in another region, the abdominal incision should be made close to the mid-line, below the umbilicus. The hand is then introduced, the obstruction sought for, and the portion of intestine involved is brought out of the wound if possible, and well walled off with gauze. If the obstruction is not readily found time will be saved by evisceration. If the stone can be dislodged it should be pushed into a portion of the bowel that is free from the inflammatory changes caused by obstruction. If impacted in the lower ileum it may be possible to push it through the ileocecal valve and to work it through the colon to the rectum, whence it can be

removed by an assistant. If enterotomy is necessary to extract the stone, the latter should be held firmly against the wall of the intestine opposite the attachment of the mesentery and a longitudinal incision made over the mass. This incision should be just large enough to allow expulsion of the calculus; and it should be made through healthy bowel, preferably on the aboral side of the obstruction. After removal of the stone the intestinal wound should be closed: (1) with a continuous through-and-through suture of catgut, special attention being paid to inclusion of the mucous membrane in the suture, and to proper inversion of the wound margins; (2) this first row of sutures should then be reinforced with a continuous sero-serous suture (Lembert or Cushing) of fine linen. If it has been impossible to dislodge the calculus it may be necessary to resect the segment of bowel obstructed; this will also be necessary if gangerne has occurred from strangulation. In most cases the abdominal wound may be closed without drainage.

The high mortality of this condition is due to delay in resorting to operation. F. Martin (1912) collected nineteen operations done within recent years, including three of his own. Of these patients eight recovered, and eleven died, a mortality of 57.8 per cent. All three of Martin's own patients recovered, though in one resection was necessary.

CHAPTER XVIII

SURGERY OF THE LIVER

Anomalies of Size, Shape, and Position of the Liver

These may be *congenital* or *acquired.* As the result of faulty development, the liver may enter the thoracic cavity, may lie immediately beneath the skin in the region of the umbilicus (hepatomphalos), may form part of the contents of a congenital umbilical hernia; or, finally, in cases of transposition of the viscera, may be located on the left side the body. In such cases the left lobe is larger than the right. Other congenital anomalies consist in absence of one or more of the ligaments; variation in the size of the lobes; or the presence of accessory lobes. In monsters the liver may be absent. Linguiform lobulation of the right lobe may be found as a congenital defect; such cases are mistaken at times for the acquired condition described by Riedel (page 535). Lobulation or other change in the configuration of the entire organ may result from prenatal syphilis, tuberculosis, or hepatitis; though it is more likely that these conditions are acquired in postnatal life.

Acquired changes in the form of the liver are found frequently. It is a plastic organ, and whenever there is prolonged abnormal pressure or traction on the liver substance, lasting changes in form are produced. The principal cause of these acquired changes in the form of the organ is found in the pressure exerted by a corset or band. In the classical monograph of Hertz (1894) two principal forms of the so-called "corset-liver" are described: In the *first,* one or both lobes are elongated downward in the form of a thin apron, which may lie over or under the intestine; and the anterior surface of the liver is marked by a transverse depression (the "corset-furrow"), the overlying peritoneum being thickened and fibrous. In the *second* form the upper portion of the liver is much thicker than the lower. The posterior surface is curved around the spinal column while the anterior surface conforms to the concavity of the anterior abdominal wall. The transverse corset-furrow is not so marked as in the first type. Mixed types may also be recognized.

The **"corset-liver"** (Fig. 153) is seen much more frequently in women than in men, although the latter are not free from it. The

change in shape is due to pressure atrophy of the liver substance, followed by the formation of cicatricial tissue. The degree of alteration in shape, and the depth of the furrow vary considerably in different cases. In some instances continued pressure will cause atrophy of practically all of the hepatic tissue in the furrow, nothing but a hinge of fibrous tissue remaining; while in others there will be a mere indentation on the convex surface of an otherwise normal liver.

The "corset-liver" is of interest to the surgeon mainly as an etiological factor in diseases of the gall-bladder and bile-ducts. This aspect of the condition was discussed at page 478.

FIG. 153.—Corset Liver from a Patient in the Episcopal Hospital Who Died from Peritonitis Due to Perforation of a Carcinomatous Ulcer of the Stomach.

Linguiform lobulation of the liver (**Riedel's lobe**) was so graphically described by Riedel in 1888, that his name has become inseparably connected with the condition. It had been recognized previously and its true significance perceived by Terrier (1888). The right lobe is the usual site of the deformity although in rare cases the quadrate, or even the left lobe of the liver has been affected. In the typical case there is no ptosis or increased mobility of the liver, but simple elongation or hypertrophy of one section of the organ. The various structures holding the liver in place are intact, and the liver maintains its normal position against the diaphragm.

Linguiform lobulation may result from tight lacing, being an extreme degree of "corset-liver;" but usually it is due to drag upon the liver by a distended gall-bladder, and in almost all cases is associated with disease of that viscus. According to Kelly enlargement of the gall-bladder has been found in 60 per cent. of all cases of lobulation.

A Riedel's lobe varies greatly in size and shape. There may be a scarcely appreciable elongation downward of the right lobe, or a freely movable, distinctly pedunculated "wandering lobe." In the latter instance the pedicle usually is attenuated, being little more than a fibrous cord which attaches the lobe to the liver. Under such conditions the

lobe will be very freely movable. Riedel's lobe is not infrequently the seat of gummata, of abscess or tumor.

According to Harris (1910), Leue in a total of 3484 autopsies found a constricted lobe in 1.9 per cent. of male subjects and in 25.3 per cent. of female subjects over sixteen years of age.

The *symptoms* of linguiform lobulation usually are those of an associated condition in the gall-bladder. There may be tenderness and pain, as a result of congestion of the anomalous lobe; or the lobe may exist for years without producing any symptoms. Distortion of the duodenum may cause dyspeptic symptoms.

Treatment.—This consists in proper treatment of any biliary lesion present. Cholecystectomy is the operation of choice; the liver usually returns to its normal shape after this operation. If no cause can be found to account for the development of the linguiform lobe, it may be excised, or sutured to the anterior abdominal wall.

Acute passive congestion of the liver, associated with cardiac incompetency, may simulate very closely an attack of acute cholecystitis. The enlarged liver is extremely tender, there is rigidity of the overlying abdominal wall, the patient suffers great pain, appears acutely ill, is nauseated, and there is leukocytosis. But the association of dyspnea and the physical examination of the heart should put the surgeon on his guard.

Hepatoptosis, movable liver or **floating liver** may be *congenital* or *acquired.*

Congenital movable liver may be due to the presence of a mesohepar, as in the case reported by Clark and Dolley (1905). Their patient was a female, thirty-four years of age, and unmarried. Her abdomen was markedly asymmetric, there being a definite bulging of the right side from the costal margin to the iliac crest. The lower lobe had no ligaments, and there was a double reflection of the peritoneum from the upper lobe to the diaphragm, producing a true meso-hepar, which measured 13 mm. in length. Albu (1909) studied ninety-four infants ranging from one to ten days old, and found visceral ptosis in 11 per cent. of the males and 44 per cent. of the females; hepatoptosis was present in 5 per cent. of the males and in 9 per cent. of the females.

Acquired movable liver, which was first noted by Heister in 1754 in his study of a cadaver, and first described in the living subject by Cantani in 1866, is not a very rare condition. Clarke and Dolley (1905) found reports of 118 cases; this included those collected by Legg, Faure, Graham, and Ssaweljew. Of these, thirteen were in men and 103 in women, and one in a child. Of the women, ninety-

three were married and ten single. In a study of 3400 patients, about equally divided as to sex, Albu (1909) found visceral ptosis in 21 per cent. of the men and 68 per cent. of the women; hepatoptosis was found in 9 per cent. of men and 17 per cent. of women.

The liver is naturally a mobile organ, moving upward and downward with the ordinary respiratory movements and also, to a slight degree, with posture. Normally, the liver is held in position by the inferior vena cava and the hepatic veins which empty into it; by the coronary ligaments and their cellulo-vascular bands; by the fibrous tissue found on the posterior extra-peritoneal surfaces; by the intra-abdominal pressure exerted by the muscles of the abdominal wall; by the so-called suspensory ligaments; and by negative intrathoracic pressure. Anything which decreases the sustaning powers of these various agencies or markedly increases the amount of work thrown upon them may be a factor in causing displacement of the organ. The causes may be divided into the following classes: (1) traumatic; (2) conditions producing increased intra-abdominal capacity, such as repeated pregnancies when associated with pendulous abdomen, removal of tumors or ascitic fluid, etc.; (3) structural increase in the size of the liver, or solid growths dragging on the organ; and (4) the use of improperly fitting corsets. Downward traction may be exerted by an abnormally shortened round ligament. Cheyne (1906) believed that "once the liver is displaced downward from the diaphragm it is sucked down still more by the atmospheric pressure and the intestines pass up between it and the diaphragm." When Heister, in 1754, found a movable liver at autopsy, he advanced the theory that the development of the condition "was due to the will of God to prove his Omnipotent power."

Landau (1885) divided cases of hepatoptosis into three grades or degrees. In the *first* grade there is moderate descent combined with version, either anterior or posterior. In the *second*, there is marked descent combined with lateral displacement toward the right and version either anterior or posterior. In the *third* there is displacement directly downward, or slightly oblique, with the left lobe usually palpable in the abdominal cavity. The liver may also be displaced upward, with rotation, so that the inferior surface will present anteriorly.

The same factors which cause changes in the position of the liver frequently have a similar effect on the other organs of the abdominal cavity. In consequence of this a general *splanchnoptosis* usually is associated with movable liver. T. R. Brown (1908) quoted Glénard

as having found, in a study of 1310 patients, fifty-one cases of hepatoptosis, thirty-two of which were associated with movable kidney.

Temporary displacement of the liver must not be confounded with movable liver, although the latter condition may be established ultimately. Such temporary displacement not infrequently is produced by pressure from pathological lesions distinct from the liver. Pressure may be exerted in a downward direction by intrathoracic conditions such as pleurisy with exudate, empyema, hydrothorax, or emphysema; by tumors or abscesses of the lungs or mediastinum; or by pathological changes between the diaphragm and the liver, such as subphrenic abscesses. Upward displacement may result from disease of the abdominal organs, especially when associated with ascites. A traumatic opening through the diaphragm may allow the liver to become involved in a diaphragmatic hernia.

The resulting degree of mobility of the liver, after the subsidence of these various affections, will be modified by the extent and duration of the pressure exerted and by the strength and abundance of the resulting adhesions.

The *symptoms* of movable liver are rather vague. In many cases the symptoms presented by the displaced liver are over-shadowed by those of the diseases causing the trouble. In all of these cases the displaced liver is of secondary importance. In movable liver pain frequently will be the first symptom noticed. This pain may begin as the result of any jarring movement of the body, or anything causing a sudden spasmodic contraction of the diaphragm. The pain may be noticed first after jumping, sneezing, coughing, etc. Later, paroxysms of pain may occur without any known cause. The pain is most commonly felt in the right hypochondrium and epigastrium but it may radiate to the right shoulder or to the right flank. The pain is relieved almost immediately by replacement of the liver in its normal position. Pressure on the liver, when ptosed, causes peculiar sensations in various parts of the body, especially in the arms and shoulders. Digestive symptoms vary. Usually symptoms of gastric dyspepsia are present; especially flatulence, discomfort during the the period of digestion, vague pains in the epigastrium and often throughout the intestinal tract, headache, insomnia, etc. There is a feeling of weight and heaviness in the epigastrium and hepatic region. Sometimes there may be found also ascites, polyuria, hemorrhoids, jaundice, recurring hemorrhages from the stomach and edema of the lower limbs. *A caput medusæ* may be found as a result of traction on the vena cava. As a rule, there is no marked dis-

turbance of the normal biliary activity, though gall-stones often are present.

The *diagnosis* of hepatoptosis is based on the symptoms mentioned, together with a demonstration of the displacement. The proptosed liver will be recognized as a large tumor in the abdomen, of the size and consistency of the liver; usually it is to the right of the umbilicus, and often a distinct notch can be felt. The tumor may be partly replaced toward its normal position where it will remain while the patient is recumbent. When it is displaced, percussion of the normal site of the liver will give a tympanitic note rather than the usual dull or flat liver note, and the pulmonary resonance posteriorly will merge with intestinal resonance. It may be distinguished from a movable or enlarged kidney by the absence of urinary symptoms; by observing that the liver moves during respiration; that it lies in front of the colon, not behind it; and by attention to the changes in the percussion note in the normal hepatic area, to which attention already has been directed. Fluoroscopic examination should be made or an *x*-ray photograph should be obtained for confirmatory evidence.

The *treatment* is palliative or operative. *Palliative treatment* consists in the application of a well-fitting binder or abdominal bandage which will retain the organ in its normal site after it has been replaced by manipulation. The pressure should always be from below upward the intestines being pushed upward to act as a cushion on which the liver may rest. The inflatable pad of Byron Robinson (1903) is a good apparatus for this purpose.

Operative treatment consists in retaining the liver in its normal position by one of the numerous methods of hepatopexy that have been devised. In replacing the liver the organ should be rotated backward and to the right, and pushed well up against the diaphragm, after examination of the space between the liver and diaphragm has shown that no coil of intestine lies between.

The first operation of hepatopexy was performed by Gérard-Marchant in 1891. He sutured the anterior edge of the liver to the costal margin with silk. Péan held the liver in its normal site by first making a transverse abdominal incision, replacing the liver, and then making a barrier below it by suturing the anterior and posterior layer of parietal peritoneum together transversely. Franke united the anterior margin of the liver, with the exception of the portion near the gall-bladder , to the costal margin and then placed gauze between the upper surface of the liver and the diaphragm. The gauze was allowed to

remain in place for eight days. Its removal was followed by the formation of strong adhesions between the liver and diaphragm. Moynihan advocated the same line of procedure, with the addition of gauze packing placed below the right lobe of the liver. Cheyne sponged the upper surface of the liver with undiluted carbolic acid. He also recommended division of the round ligament when that structure seems too short. In all forms of operation the patient must remain on the back in bed for at least four weeks, at absolute rest. Elevation of the foot of the bed a few inches will also aid in overcoming any tendency of the liver to prolapse during the formation of strong adhesions. For some months after operation a well-fitting abdominal belt or binder should be worn and all violent efforts should be carefully avoided.

Abscess of the Liver

Abscess of the liver (**suppurative hepatitis**) may be classified in various ways: as *single* or *multiple;* as *tropical* (amebic) or *non-tropical;* as *primary* (traumatic) or *secondary;* according to the *character of the infecting micro-organism;* or according to its *location*, the "anatomical" classification.

Probably the best classification is that which recognizes 1. **Traumatic abscess,** in which there is a wound or contusion of the liver substance followed by infection from the exterior or from the blood. 2. **Pyemic** (**embolic abscess,**) in which infection reaches the liver by means of infected emboli, through extension of suppurative processes adjacent to the liver, by direct infection, etc. 3. **Amebic** (**tropical**) **abscess,** in which the ameba coli is the chief etiological factor. Rarer forms of abscess in which the liver becomes honey-combed with cavities containing pus and varying in size from a pea to a walnut, sometimes larger, are due to **tuberculosis,** or **actinomycosis** Suppuration also frequently occurs in an **hydatid cyst** (page 559).

Etiology. Predisposing Causes.—*Traumatic abscess,* as its name implies always is preceded by a wound or contusion involving the integrity of the liver cells. In cases of penetrating wound, by stab or gunshot injury, or when the liver is punctured by the fragment of a rib in compound fracture, the infection is admitted from without. In cases of contusion, or subcapsular rupture of the liver, the hematoma which forms may be converted into an abscess by infection through the blood, or lymphatics, or even through the biliary tract (page 545). *Pyemic abscess* may have as a predisposing cause any suppurative, infectious, or parasitic condition in any portion of the body. Espe-

cially frequent are lesions in the distribution of the portal vein, such as **appendicitis** or **typhoid fever.**

Melchior (1910) gave the following figures showing the frequency with which *typhoid fever* is complicated by hepatic abscess.

Author	Cases of liver abscess	Cases of typhoid fever	Percentage
Holscher	12	2000	0.6
Horton-Smith	2	....	0.58
Piorkowsky	2	1229	0.16
Vierhuffs	3	1186	0.25
Berg	0	1662	....

The hepatic abscess usually develops during convalescence from typhoid fever, but a year has elapsed in some cases. The average fever-free interval is fourteen days—the period between defervescence from typhoid fever and the development of symptoms from the hepatic abscess. The infection may reach the liver through the bile-ducts, through the systemic circulation, or by way of the portal vein. Four cases, all fatal, followed appendicitis of typhoidal origin. Melchior collects records of twenty-five cases, in none of which no operation was done. Of these nine patients only two recovered, the abscess in the first case (1869) discharging spontaneously through the lung, and that of the second pateint (1875) rupturing through the bowels. In four cases no formal operation was done, the abscess merely being punctured; all of these patients died. Of twelve patients in whom the abscess was treated by incision, in the ordinary way, only two died, a mortality of 17 per cent. This clearly demonstrated that the proper treatment of hepatic abscess in typhoid fever is by operation, according to the usual technique (Chapter XXIV).

As regards *hepatic abscess of appendicular origin*, it usually has been taught that the foci of suppuration in the liver are multiple and widespread; and there is no doubt that this is so in most cases, and that the condition is exceedingly fatal. Personally we never have seen recovery occur in a case of hepatic abscess secondary to appendicitis; but such cases have been reported in a few instances. Quénu and Mathieu (1911) note that although foci of suppuration in almost all these cases are multiple, yet that some patients have had only one or at most two solitary abscesses, and that in other cases the abscesses, even if multiple, are closely congregated in the right lobe of the liver

and should be amenable to operative treatment. Loison, in 1900, reported twelve such cases, all the pateints having died without operation because the possibility of cure by operation was not recognized. Loison reported one patient under his own care, whose life was saved by timely operation, and he says that Körte in 1892 also had one patient who recovered after operation. We are inclined, however, to agree with Tuffier, who in the discussion which followed the reading of Loison's report said that to him the diagnosis in Loison's patient seemed uncertain; it might have been a case of subphrenic abscess following appendicitis. Quénu and Mathieu collected records of fourteen operations for this complication of appendicitis, with only two deaths. The cases suitable for surgical intervention are not those presenting the usual picture of diffuse suppurative hepatitis or pylephlebitis closely following the attack of appendicitis, but those in which the symptoms of liver abscess develop after a free interval, and in which the physical signs indicate that a single abscess may be present. The operative methods in these cases are the same as in cases of tropical abscess of the liver (p. 791).

Abscesses as a result of suppurative processes in the structures adjacent to the liver are not common. They may follow acute empyema of the gall-bladder, perforation occurring through the portion of that viscus which is in contact with the liver substance. Direct extension from diseased conditions of the pylorus and pancreas, after the formation of adhesions binding these structures to the liver, may result in infection and abscess formation in the liver; but this is rare. Liver abscess due to *suppurative cholangeitis* almost invariably is multiple and a sequel of lesions of the biliary apparatus (Plate VII). Theoretically it is possible for such intestinal affections as typhoid fever, gastroduodenitis, duodenal ulcer, etc., to infect the bile-duct by continuity of structures, but in almost all such cases it is quite evident that the foci of suppuration in the liver are due to septic emboli received through the portal system or by way of the systemic circulation, as in pyemia.

Liver abscess has been shown to follow *influenza, yellow fever* and many other *infectious diseases.* Suppurative venous thrombosis with the production of septic emboli and abscess of the liver may follow almost any infective process in the body either by way of the portal or the systemic circulation. These predisposing causes were well summarized by Munro (1905), as follows: "Malaria, infections of the thoracic organs or of the umbilicus, pyemia arising from various infections of any portion of the body, anthrax, ulcers of the intestinal tract, pelvic infections, splenic abscess, abscess of the mesenteric lymph-

nodes, infections of the biliary tract and pancreas, from peritonitis, echinoccoccus, infection after hemorrhoidal operations, etc."

McWilliams (1907) in an analysis of sixteen cases of abscess of the liver found that ten of them exhibited no apparent or evident etiological factor.

Climate.—Climate plays a very important part as a predisposing cause of amebic, or tropical, abscess. It is a change of climate, however, rather than the climate itself which is the important factor. Tropical dysentery, which is so frequently the forerunner of abscess of the liver, occurs most frequently among those who go to the tropics from a temperate climate; and especially among those who do not conform to the diet and customs of the tropics. Herrick (1910) from a study of abscess of the liver in the Panama Canal Zone, estimated that the white man from the United States is the most susceptible of all laborers, and that the colored employees from the islands are the least susceptible. He found one case in every 1178 whites from the United States; one case in every 2240 whites from Europe; and one case in every 3722 colored employees. Kieffer (1903) studied thirty-three cases of liver abscess among the soldiers in the Phillippines, and found a history of dysentery in each; among twenty-five cases of abscess among the natives and civilians there was a history of dysentery in twenty-two. Keiffer concluded from his investigations that 20 to 25 per cent. of the cases of severe amebic dysentery result in liver abscess; and that 85 per cent. of tropical abscesses are due to the ameba.

The frequency of tropical abscess as a result of dysentery varies with the statistics of different countries. In India, about 35 per cent. of the soldiers who died from dysentery had pus in the liver, while liver abscess was found in less than 5 per cent. of the patients in the Philippines who suffered from dysentery (Rhoads, 1904). It is safe to assume that all cases of amebic abscess of the liver have been preceded by a dysenteric ulceration of the intestine. In those cases where causal relation cannot be determined, either the symptoms of dysentery were so slight that the patient forgot all about them, or the ulcerations were slight and the condition ran a latent course without the production of marked symptoms.

Coffin (1906) found 1523 cases of dysentery among 10,603 patients treated in the United States Army Division Hospital in Manila in three and one-half years. Of these, 859 were of the amebic type; 236 were of the catarrhal type; and in 428 cases the type was not noted. The majority of cases were sent home invalided, in from one to eight

weeks, this fact accounting for the small number of liver abscesses found, thirty-four.

The disease is particularly prevalent in the Philippine Islands, India, and Egypt.

Exciting Causes.—All abscesses of the liver are the result of bacterial or parasitic invasion. Foreign bodies, such as fish-bones, pieces of straw, etc., have been found in the contents of the abscess cavity, but it is probable that the abscess resulted from the presence of micro-organisms and was not caused by the foreign body alone. Coccidia and the ray fungus of actinomycosis have been underlying factors; in rare instances the bacillus of tuberculosis may act as the direct causative factor.

Bacteria may gain entrance to the liver through a wound communicating with the exterior of the body; through extension of suppurative conditions in the adjacent structures; through the portal vein, the hepatic artery, the hepatic vein, or the lymphatics from adjacent or far distant points of infection; or through the biliary passages. The most frequent pathway of entrance is through the portal vein, the radicals of which carry the infection from more or less distant foci. In other than tropical climes the most frequent source of infection is found in disease of the appendix; in tropical countries the infection most frequently is a sequel of dysentery.

The micro-organisms which are found most frequently in liver abscesses are the Amœba coli, the B. coli, the streptococcus, the B. pyocyaneus, the B. dysenteriæ, the D. pneumoniæ, the Actinomyces bovi, the S. pyogenes aureus and albus, and B. typhosus.

Pathology.—Liver abscess may be solitary or multiple. Traumatic and amebic abscesses usually are single; pyemic (embolic) and tuberculous abscesses usually are multiple. The majority of abscesses develop in the right lobe of the liver, and usually are nearer the convex than the concave surface. They vary greatly in size, from minute foci to collections of pus that may occupy the greater part of the entire organ.

The pathological changes found in the liver vary with the underlying etiological factors causing the abscess. The contents of the abscess may vary in color from yellow to green or brown; in almost all cases microscopical study reveals the presence of bacteria in addition to liver cells and detritus, which are always present. The bacteria usually found are streptococci, staphylococci and colon bacilli. The walls of the abscess cavity are irregular and are composed of infiltrated liver substance.

Traumatic abscess always is septic. When there is a subparietal injury, the bacteria of suppuration may be introduced into the devitalized portion of the liver either through the bile-channels, the portal vein, the hepatic artery or the hepatic veins, or directly from the liver substance where there has been previous disease of the organ. When there is an external communication with the liver the germs of suppuration usually are introduced through this channel, frequently as a result of unnecessary probing and examination of the wound. The site of an abscess following trauma is determined by the site of the injury. The size varies with the extent of the injury and the virulence of the infecting micro-organism.

In embolic or pyemic abscesses, which comprise those usually seen in temperate climes, the process is somewhat different from that found in the traumatic abscess. Emboli carrying germs, lodge in one or more of the terminations of the portal vein, with resulting congestion, liquefaction and purulent degeneration of the liver substance. In many instances the emboli may be carried to the liver through the hepatic artery, the emboli passing through the capillaries of the lung, entering the left side of the heart, and then being carried with the arterial stream to the liver or other organs where abscesses are formed. It is probable that actual emboli do not pass through the pulmonary capillaries, but that clumps of bacteria are carried into the systemic circulation, and that these, when they are arrested in the next set of capillaries, or when they reach a situation favorable to their growth, there induce thrombosis and suppuration.

Pyemic (embolic) abscesses usually are multiple and may occupy any portion of the liver substance, although they are found most frequently in the right lobe. They generally are near the surface of the liver and vary in size from minute foci of suppuration to masses the size of an entire lobe. Small foci frequently coalesce to form large abscesses. Microscopical examination of the contents of embolic abscesses shows the presence of active bacteria in all instances. The liver substance surrounding the suppurating process always shows inflammatory changes.

In the *tropical* or *amebic liver abscess*, the destructive process is said to begin in the hepatic cells. This process sometimes begins immediately after the onset of amebic dysentery; but in most cases weeks, months, or even years elapse before the destructive process is manifest. Musgrave and Clegg (1904) established the fact that amebæ are amenable to changes in environment and that the power to propagate in temperate climes depends on the similarity to the old environment, and

possibly on their ability to produce lesions in the tissues. The development of the abscess may be very slow.

Purulent softening causes coalescence of adjacent acini until the destructive process may involve an entire lobe of the liver. The abscesses are solitary in about 60 per cent. of the cases, and are most frequently found in the right lobe. Among 240 cases studied by Waring, the right lobe was affected in 163. Rouis (1860), in an analysis of 156 cases, found the abscess in the right lobe in 122; and Musgrave and Clegg (1904) found the right lobe involved in 95 per cent. of their cases. Langenbuch (1894) explained this preference for the right lobe by the distribution of the branches of the portal vein. The branch going to the right lobe is larger than that to the left, and runs parallel to the course of the vein, while that to the left lobe runs at right angles. The separate currents in the portal vein were alluded to at page 42; the fact that most of the usual foci of infection are in the area which drains into the right lobe is another reason for the frequency with which this portion of the liver is affected.

When the abscess is within the right lobe the liver may be normal on inspection even if the abscess is large. Distinct elevation will be noticed as the abscess nears the surface. Peritoneal irritation usually is followed by the formation of adhesions between the liver and the abdominal wall, the adjacent viscera, or the diaphragm. In many cases some evidences of pleuritis are found.

Amebic abscesses vary in size from minute foci of pus to a collection the size of the liver itself; almost the entire liver may be converted into a sac, and as much as 8000 c.c. have been removed from such a sac.

The contents of the amebic abscess vary with the duration of the infection and with the extent of the destructive process. The consistency varies from fluid to gelatinous. The color usually is brown. As a rule, bile is not a constituent of the abscess, the contents being composed almost exclusively of liver cells and detritus. In cases of very long duration, yellow pus may be found.

Microscopical examination usually reveals amebæ, especially in specimens obtained from the walls of the abscess. Kieffer (1903) was able to demonstrate neither bacteria nor amebæ in 20 per cent. of his cases; in 60 per cent. bacteria were present at the time of his examination. The amebæ are demonstrable in the majority of cases, although it may be necessary to wait until there has been a discharge from the wound for three or four days before they can be found.

The wall of the abscess consists of liver substance which is soft and

irregular and, exhibits masses of necrotic liver tissue. Surrounding the abscess wall may be found areas of necrosis which usually do not present signs of suppuration.

Symptomatology.—The symptoms of abscess of the liver vary with the nature and severity of the preexisting condition, with the pathway of infection, and with the nature and virulence of the infecting micro-organism. In a great many instances, the symptoms are over-shadowed by those of a coexisting inflammatory disease, especially of the biliary tract. In other instances the abscess is latent, giving rise to no symptoms, and being found unexpectedly at autopsy.

In *traumatic abscess* there usually is pain in the liver region which is markedly increased by pressure. Change of position will often cause increase in pain. If the abscess be located in the upper part of the right lobe pain will often be referred to the shoulder. A palpable friction rub may be elicited in some cases where perihepatitis is present. If there is involvement of, or pressure upon, the biliary ducts, jaundice will be present. The liver usually is enlarged, more or less irregular in outline, and a fluctuating mass possibly may be detected. Chills, fever, sweating, and a high leukocytosis generally are present. If an external wound allows the discharge of pus, hepatic cells will be found in the pus or in the scrapings.

In *pyemic* (*embolic*) *abscess* the symptoms may be entirely overshadowed by the symptoms of the primary disorder. As a rule the hepatic symptoms, if any are noticed, develop while the primary affection, such as appendicitis, is still active. When the liver complication remains latent for a time, as sometimes is the case according to Quénu and Mathieu (1911) when the infection follows acute appendicitis, the symptoms may not become evident until weeks after the entire disappearance of all appendiceal symptoms (page 542). In these cases the general condition of the patient may not be satisfactory and possibly there may be a persistent pallor with emaciation, but a comparatively long free interval will elapse before the symptoms of acute suppurative hepatitis assert themselves. In such cases the course resembles that seen in amebic abscess. In the usual type of case, the most significant local symptoms are *pain* in the *hepatic* region, often referred to the right shoulder, and *tenderness* of the liver with *enlargement* of that organ. The general symptoms consist of *chills*, *sweats*, *fever* of the remittent type, perhaps *jaundice*, and a marked *increase in the leukocytes*.

Through the courtesy of Dr. Homer C. Bloom, with whom he was associated, the senior author is able to report the following case:

E. B., fifty-three years of age; native of France; had had frequent attacks of what he said his doctors called gall-stone colic; these attacks dated back ten years; had been jaundiced more or less ever since the first attack; drank rather heavily of alcoholic stimulants; was a large eater. Had been losing strength and flesh rapidly during the past year, more or less gastro-intestinal disturbance, marked constipation.

April 22, 1905, patient suffering severe and agonizing pain over the region of the gall-bladder; there was marked tenderness over this region as well as over the upper part of the abdomen and liver. Jaundice almost bronze in color. The attack had been ushered in by a chill. There was marked dullness over the entire right side, extending three inches below the lower rib and as high as the right nipple and posteriorly to the level of the angle of the scapula. There was considerable distention of the abdomen and pronounced rigidity over the region of the gall-bladder.

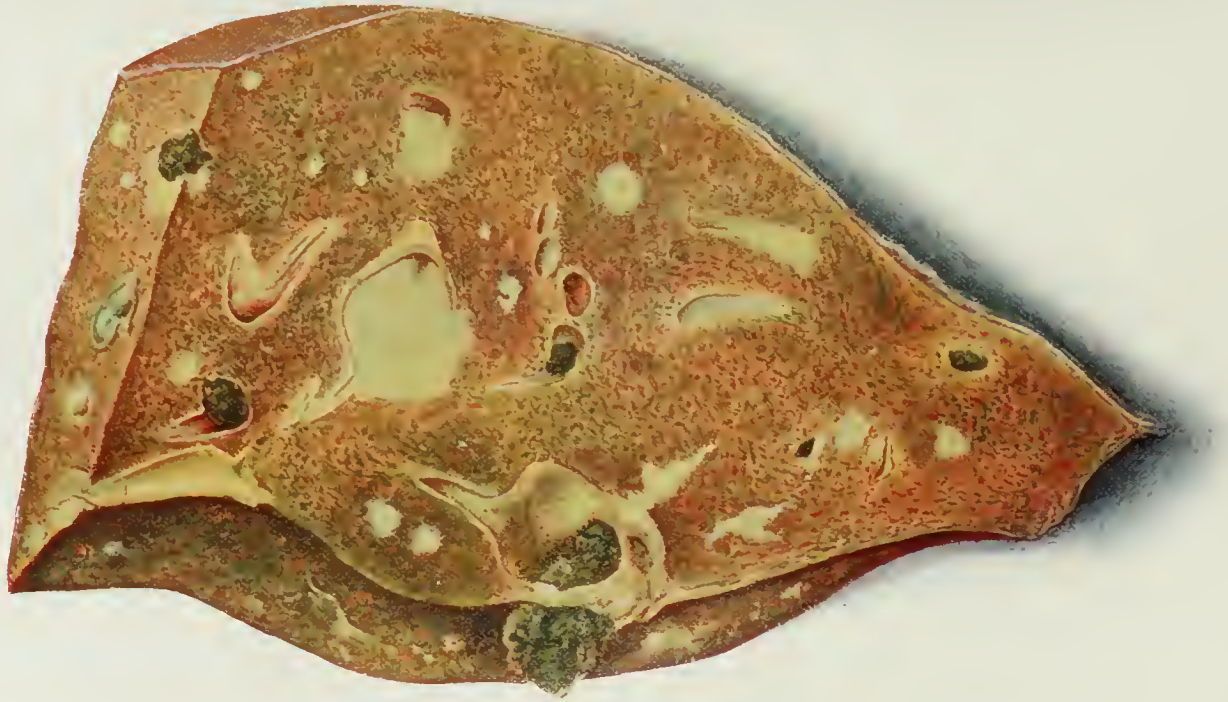
Attacks continued at longer and shorter intervals until May 8, when operation was performed.

Operation.—Common duct distended with small stones and débris; common duct opened and emptied, but immediately from the liver the same character of material escaped in large amounts. A drainage tube was put in the common duct and the same material with bile continued to drain until the death of the patient.

Autopsy revealed a tremendous liver at least three times the weight of a normal liver; all through the structure of the liver were minute abscesses and in that part nearest the ducts were hundreds of biliary stones the size of a small pea. The kidneys were diseased, the right side of the heart dilated and the abdominal cavity filled with bloody fluid. A section of the liver is shown in Plate VII.

In *amebic (tropical) abscess* the symptoms, in 33 per cent. of the cases, are entirely latent, the abscess either remaining quiescent or growing so insidiously that rupture gives rise to the first demonstrable symptoms (Rouis). Spontaneous rupture is a common event in large abscesses, Cyr (1887) having found a rupture in 159 cases in a series of 563, or in about 28 per cent. The abscess opened into the lungs in fifty-nine, into the pleura in thirty-one, into the pericardium in one, into the peritoneum in thirty-nine, into the intestine in thirteen, into the stomach in eight, into the kidney in two cases. In the great majority of all cases (nearly 60 per cent.) rupture occurs through the diaphragm. Flexner (1897) reported two instances of rupture into the inferior vena cava.

The onset of the amebic abscess usually is slow. The patient for months may complain of general ill-health, malaise and increasing weakness, without evincing any symptoms that refer directly to the liver. When there is a previous history of life in the tropics, of dysentery or other marked intestinal disturbance, these general symptoms become significant and a careful examination of the hepatic region and of the stools should be made. Pain is entirely absent in about



Suppurative Cholangeitis with Multiple Abscess of the Liver. (Case History, page 548.)

Face p. 548

PLATE VII

20 per cent. of cases. In other cases there will be a dragging sensation and some discomfort. When the abscess approaches the peritoneum, there will be actual pain, at times sharp and stabbing. The pain seldom is referred to the shoulder, unless the abscess is near the surface, near the gall-bladder, or in the Spigelian lobe. The temperature may be normal or of a severe septic type, depending upon the presence or absence of pyogenic bacteria in the abscess. In the purely amebic cases, there is very slight increase of leukocytes with a corresponding increase of polynuclears. Jaundice will not be present unless there is pressure on the larger branches of the hepatic ducts.

Diagnosis.—In typical cases a diagnosis usually is not difficult. In other cases, either because the symptoms vary so greatly from the type, or because they are absent, a diagnosis may be very difficult to make. A provisional diagnosis should be made from the history of the case, the general appearance of the patient, and the clinical picture presented. If the patient's physical condition is not very serious, further study generally will make the diagnosis clear. Exploration of the liver with a trocar never should be practised on account of the great danger of infecting either the peritoneal or the pleural cavity. If exploration is necessary to establish a diagnosis, an incision should be made of sufficient size to expose the liver and permit its examination by the fingers and the exploring needle under full control of vision.

Skiagraphic examination is of value in showing the extent of the swelling of the liver, and in determining its upper border. It is of little value in arriving at a differential diagnosis, but may be of great aid in determining the size, position, and mobility of the liver.

In *traumatic abscess*, where a communication leads from the exterior to the abscess cavity, the diagnosis may be cleared up at once by finding liver cells in the pus being discharged. Where there is no external wound, the diagnosis depends on the history of traumatism followed by symptoms which point to a lesion of the liver accompanied by fever of a septic type, chills, etc.

A mistaken diagnosis may be made, as is shown in the following case.

A. M., male, aged forty-two years, admitted to the German Hospital December 3, 1912. In 1893 the patient had been in the British Army in Egypt and had had a severe attack of dysentery. During the four years he remained in Egypt he had had repeated attacks of dysentery and lost considerable weight. Since leaving Egypt, his bowels had been regular, but were moderately loose at times although there was no return of the dysentery.

Five months prior to admission to the hospital, he was kicked in the abdomen by a horse and was in bed from the effects of the injury five weeks. He then returned to work but had to stop and return to bed. Vomiting began soon after the reception of the injury and has been more or less persistent ever since. He has lost ten pounds in weight. Has not been jaundiced at any time. Two days before admission, patient had a severe chill which lasted for two hours.

On admission, examination revealed a mass the size of a grape fruit centering in the gall-bladder region, somewhat nodular and evidently part of the liver. It extended below the ribs in the mid-costal line. The mass was tender. Abdomen otherwise negative. Diagnosis of liver abscess made.

Operation, December 7, 1912.—Ether anesthesia. Incision through right rectus muscle. Examination of the mass proved it to be located entirely within the gastro-hepatic omentum, the liver being apparently normal. A trocar and canula introduced into the mass and a considerable quantity of dark fluid aspirated No rupture of the common duct detected. Cavity drained and wound closed to drainage. Examination of the aspirated fluid showed it to be blood, and free from bile. Patient recovered, and left the hospital January 13, 1913.

In *pyemic (embolic) abscesses* the diagnosis cannot be made readily if the symptoms of the primary disorder are at all prominent. The general symptoms of involvement of the liver during, or following, any acute infectious disease or any suppurative process should make the diagnosis of abscess probable. Usually there will be fever, chills, sweats, marked increase of leukocytes, and sometimes jaundice, enlargement of the liver, with pain and tenderness beneath the right costal margin.

In *amebic (tropical) abscess*, the diagnosis often is attended by much difficulty. Here the past history is most important. With a history of dysentery or a sojourn in tropical climes and the presence of pain in the region of the liver, often referred to the right shoulder, tenderness and progressive enlargement of the liver, leukocytosis, especially if increased in the evening, fever, chills and sweating, the diagnosis of amebic abscess may be made. The fever will vary from a very mild to a moderate or a severe degree, depending upon the presence or absence of pus-forming micro-organisms. When the amebic abscess becomes secondarily infected with pyogenic bacteria the symptoms will be much more severe than in the simple amebic cases. The leukocytes will vary from 15,000 to 30,000, part of which increase may be due to the intestinal disease. According to McDill (1907), colonic irrigations and saline purgatives will eliminate the leukocytosis due to the intestinal infection. One of the best indicators of the presence of pus is a high leukocyte count in the afternoon. Examination of ordinary stools may be negative. McDill claims, however, that if amebæ are present they always will be found in the third or fourth watery stool

following the administration of saline purges. It must be remembered that the amebic abscess may be latent and may remain so for an indefinite period. Manson states that "the most common mistakes in diagnosis are: (1) Failure to recognize the presence of disease of any description, even when an enormous abscess may occupy the liver. (2) Misinterpretation of the significance and nature of a basic pneumonia, a condition so often accompanying suppurative hepatitis. (3) Attributing the fever symptomatic of liver abscess to malaria. (4) Mistaking other diseases for abscess of the liver and *vice versa*—for example, hepatitis of a non-suppurative nature, such as that attending malarial attacks; suppurative hepatitis before the formation of abscess; syphilitic disease of the liver—softening gummata which are often attended with fever of hectic type; pylephlebitis; suppurating hydatid; gall-stone and inflammation of the gall-bladder; subphrenic abscess; abscess of the abdominal or thoracic wall; pleurisy; encysted empyema; pyelitis of the right kidney; pernicious anæmia; leukocythæmia; scurvy and other similar blood diseases associatd with enlargement of the liver; ulcerative endocarditis; kala-azar; Malta fever; trypanosomiasis. Any of these may be attended with fever of hectic type, increased area of hepatic percussion dullness, and pain in and about the liver."

In malaria the spleen is proportionately larger than the liver. In cases of amebic abscess of the liver the spleen is not enlarged. If the spleen is enlarged in a case of hepatic abscess, this probably is embolic in origin. The fever in malaria is marked during the day, while in liver abscess the rise of temperature is seen in the evening. The blood picture in malaria is quite different from that of hepatic abscess, the former showing the plasmodium which is absent in the latter; in abscess there usually is a high leukocyte count with a corresponding increase of the polynuclear cells, which is not seen in malaria. The administration of quinine will generally clear up the diagnosis between malaria and liver abscess.

In cholelithiasis and cholecystitis and in chronic calculous obstruction of the common duct, the history will have great weight in making the diagnosis. In these conditions the symptoms point to conditions of the biliary tract rather than to the liver itself. The pyemic abscess, complicating acute infection of the biliary tract, cannot always be recognized, although the increased area of tenderness over the hepatic region in liver abscess might cause one to infer that the liver had been infected.

Prognosis.—The prognosis in *traumatic abscess* will depend upon the character of the injuring force, the portion of the liver involved, and the

virulence of the infecting mico-organism. The prognosis always is grave.

In *pyemic abscess*, especially when multiple, the prognosis always is very grave and according to some writers, hopeless. Dieulafoy stated (1900) that infection of the liver from acute appendicitis is always fatal. His statement was challenged by Quénu and Mathieu (1911) who analyzed fourteen cases which they claim are of this nature. The infectious focus was single, or there were only few foci, and operative measures cured twelve of the fourteen cases (page 541). When the foci are multiple the condition almost always results fatally. Personally, we have never seen recovery in a case of multiple abscess of the liver due to acute appendicitis.

In *amebic abscess* death may occur in untreated cases from sepsis following perforation into the peritoneal cavity with peritonitis. Spontaneous cure may result if the rupture occurs into the pleural cavity or the lung, but very rarely if the rupture drains the abscess into the stomach or colon. Early operative interference always makes the prognosis more favorable. Coffin quotes Manson as having found a mortality of 57.7 per cent. in the Indian Army from 1891 to 1894. Of thirty-four cases analyzed by Coffin, sixteen died. One patient died out of three operated upon by the senior author. Herrick (1910) lays great stress on the differential leukocyte count, in making the prognosis of these cases. He found that when the polynuclear count was below 80 per cent. the prognosis was good, the operative mortality being only 6.6 per cent. whereas in cases where the polynuclear cells were over 80 per cent. the operative mortality was 38 per cent.

Treatment.—The proper treatment of liver abscess is drainage. As pointed out by Herrick "the one vital necessity is that the abscess should be opened at its point of election, which would be the point where the abscess approaches nearest to the surface of the liver."

Two avenues of approach are open to the surgeon: one through the peritoneal cavity, and the other the transpleural route. The "combined operation," similar to that described in connection with injuries of the diaphragm (page 285), presents no advantages, and should not be employed. No attempt should be made to localize the abscess by means of the aspirating needle, as it is unreliable and fatalities have followed its use.[1] If the abscess cannot be clearly localized by the physical findings, an exploratory laparotomy should be performed. This will enable the

[1] Puncture of the liver was long in use as a therapeutic measure under the name of "hepatic phlebotomy" (Harley, 1886).

surgeon to make a careful examination of the liver, and show him where the incision into the liver should be made. If accessible from within the abdominal cavity, the abscess may now be opened, after isolating the field of operation by gauze packs. In most cases, however, the approach by thoracotomy is to be preferred. If the needle is used for exploration it should be followed by immediate operation, before withdrawing it, whenever pus is found. However, we are firmly of the opinion that it is dangerous to explore before sufficient exposure has been obtained by a free incision. If the abscess cannot be located readily after the abdomen has been opened, it may then be proper to resort to puncture of the liver with an exploring needle. Under such circumstances, as pointed out by Terrier and Auvray (1907), this procedure can do no harm, and may do good, by relieving the hepatic congestion by means of what Harley (1886) called *hepatic phlebotomy*.

The transpleural route, employed in 1885 by Knowsley Thornton, gives the best access to the greatest area and should always be used unless the abscess presents anteriorly, or unless laparotomy is indicated for diagnostic purposes. (For description of operation see Chapter XXIV). The transpleural route was employed by Israel, as early as 1879, in a case of hydatid cyst of the liver (page 567).

Cirrhosis of the Liver

Two main types of cirrhosis of the liver are recognized, *portal* and *biliary*. Kelly (1908) used these terms, with subtypes, because in one the etiological factor is "perhaps always transmitted by the portal circulation, the new-formed connective tissue is especially conspicuous in and about the portal spaces in the liver, and the obtrusive symptoms are those of portal obstruction." The other is called biliary cirrhosis because the essential lesion is a radicular cholangeitis and the conspicuous clinical feature is jaundice, due to obstruction to the free flow of bile.

In *portal cirrhosis* the obtrusive symptoms are ascites and gastrointestinal hemorrhages, the latter being the result of varices in the ultimate branches of the portal system of veins. It was maintained by Hale White, Rolleston, and others and Kelly concurred in this view, that patients with uncomplicated portal cirrhosis of the liver do not long survive the onset of ascites, rarely living long enough for more than one tapping to be necessary. These authorities claim that in patients who live and are tapped many times, either the diagnosis of portal cirrhosis is incorrect, or the condition is complicated by peri-

hepatitis, chronic peritonitis, etc. It is only in this latter class of patients that the question of surgical treatment arises.

In cases of *biliary cirrhosis* there sometimes are accompanying lesions of the gall-bladder and bile-ducts, which may call for surgical treatment. A distinction was drawn by Kelly between true biliary cirrhosis so complicated, and cases of obstructive jaundice with chronic intrahepatic pericholangeitis, resulting in disseminated cirrhosis of the liver. But W. J. Mayo (1918) states that he has never seen a case of biliary cirrhosis which could be considered such as used to be known by the name of Hanot (1895) and he is of the opinion that such a disease probably does not exist. The large majority of cases periously so-called he classes as instances either of hemolytic icterus or the ordinary type of biliary cirrhosis; the former is primarily a splenic disease with a work hypertrophy of the liver, but as in 60 per cent. of the cases in which he has removed the spleen for this disease there were also present gall-stones with recurring exacerbations of infection, the confusion is not hard to understand. Eppinger and Ranzi, as long ago as 1914, stated that in all cases of "hypertrophic cirrhosis" with jaundice and enlarged spleen, but with no alcoholic history, splenectomy must be considered. They had done 10 splenectomies in this class of cases, with 2 deaths; one patient was not traced, but seven remained in good health at periods varying from 4 months to 2 years after operation.

Surgical treatment in cases of *portal cirrhosis* of the liver had, until recently, for its object the relief of ascites, or of recurring hemorrhages, and not the cure of the lesion in the liver. The operations formerly advocated, therefore, must be considered as measures taken for the relief of symptoms and not as therapeutic measures inaugurated to cure the underlying disease. Little can be expected from operative treatment of cases of *obstructive jaundice* if this is postponed until hepatic cirrhosis has developed. *Surgical treatment of hepatic cirrhosis therefore is confined almost exclusively to relief of the ascites or hemorrhages which accompany the portal form of the disease.*

Etiological Factors.—It usually has been taught, on the authority of Rolleston, that ascites in cases of cirrhosis of the liver is due to portal pressure and to toxemia, the former causing conditions favorable to peritoneal effusion, add the latter interfering with the normal activity of the endothelial cells of the peritoneum. Portal obstruction alone is not the chief factor in the production of ascites, because the radicles of the portal vein lie nearer the mucous than the serous surfaces of the gastro-intestinal tract and dilatation of these radicles results in the production of varicosities; and these varicosities manifest their pres-

ence not by the occurrence of ascites, but by hemorrhages. Ascites is caused almost solely by changes in the endothelium composing the peritoneum; it is in the nature of a chronic serositis. In other words the toxemia due to disordered hepatic function is a much more important cause of ascites than is the existence of portal obstruction. *But cases of portal cirrhosis sometimes are complicated by tuberculosis of the peritoneum, or by a chronic polyserositis associated with cardiac disease;* and in such cases it may not be the hepatic toxemia but the complicating disease, which is responsible for the peritoneal effusion.

Under normal conditions the various anastomoses between the portal and systemic venous channels are able to care for the slight obstruction of the portal system. As stated by Deaver, "the radicles of the portal vein anastomose with the systemic veins in numerous places; among these anastomoses are the following: Radicles of the superior hemorrhoidal vein anastomose with branches of the middle and inferior hemorrhoidal veins, these last being tributaries of the internal iliac vein; the gastric tributaries of the portal vein anastomose with the lower esophageal veins, which empty into the azygos vein. In the suspensory ligament of the liver are veins which connect the portal system with the veins of the diaphragm; along the round ligament of the liver there are one or two veins which effect an anastomosis between the portal system and the veins of the abdominal wall."

When there is obstruction of the portal system, as is seen in cirrhosis of the liver, these anastomosing channels become enlarged to assist in establishing collateral circulation. The enlargement of the spleen, so frequently seen in connection with cirrhosis of the liver, is due to the damming back of the blood in the splenic vein as a result of the interference with the portal circulation. If the obstruction of the portal system cannot be cared for by the enlarged venous anastomoses, leakage occurs with repeated hemorrhages from the gastro-intestinal tract.

Most of the operative methods proposed for the treatment of ascites with cirrhosis of the liver seem to have been based on the theory that the ascites occurred as a direct transudate from the obstructed portal system. Many of the methods therefore are irrational, and if successful the happy issues have been due to factors not recognized at the time as essential. Vidal has well pointed out that efforts to relieve the ascites by attempts to establish a collateral circulation between the portal and systemic circulations are based on an erroneous idea of the pathogenesis of ascites. The quickest and surest way to establish such a collateral circulation is to make an anastomosis between the portal vein and

the vena cava (Eck's fistula) thus draining the portal blood directly into the right heart. This delicate operation was successfully performed by Vidal (1903); he was forced to adopt it because he found there was no omentum available for epiplopexy, and as the patient was nearly exsanguinated from repeated hemorrhages, some method of relieving the portal congestion seemed imperative; but, though the hemorrhages were cured the Eck fistula did not prevent the recurrence of ascites, six weeks before death, which occurred four months after operation, and was due to an acute general infection evidently enterogenous. The liver, interposed as a filter to the hordes of microbes constantly absorbed by the portal blood stream, was side-tracked by the operation and these bacteria entered the general circulation with undiminished virulence, so that death from acute general infection must always be anticipated under such circumstances. Another reason for condemning the operation, emphasized by Vidal, is that the withdrawal of the functions of the liver from the digestive and metabolic processes, necessitates an almost impossible restriction of diet.

The various methods of operative treatment proposed for cirrhosis of the liver have been summarized by Ricketts (1909), as follows:

1. Incision through the abdominal wall, with temporary or permanent drainage of the ascitic fluid. A number of cases have been permanently benefitted by this procedure.

2. Paracentesis, or puncture through the abdominal wall, with temporary or permanent drainage. A number of recoveries have been reported, usually after a number of tappings. Lecreuz reported, in 1902, the history of a patient from whom he removed, during a period of five years, 1750 liters of fluid by sixty-five punctures; the patient remained well two years after the last tapping. The procedure, however, usually is useless except for temporary relief of the distress resulting from the pressure of the ascitic fluid.

3. Hepatotomy, or incising the liver to various depths after having first opened the abdomen, with temporary or permanent drainage.

4. Hepatotomy with a trocar, followed by temporary or permanent drainage. The results obtained are about the same as with hepatotomy with the knife.

5. Cholangeiostomy. Thornton in 1887 succeeded in draining the biliary tract by penetrating the right lobe of the liver to one of the larger branches of the hepatic duct, in which calculi were lodged.

6. Cholecystenterostomy has been performed by Combenale and Dubar for the purpose of effecting better drainage.

7. Cholecystostomy was performed by Delagenière in 1901 for cirrhosis and this method of treatment was fully discussed by his pupil Bernard.

8. Injection of caustics into the peritoneal cavity has been resorted to on the theory that the irritation produced will cause increase in the activity of the peritoneum.

9. Ligation of the portal vein has been performed for the purpose of lessening the congestion of the liver and encouraging the establishment of a collateral circulation. The procedure was described by Pascale in 1901.

10. Hepatopexy (page 539) was performed by Delagenière in 1897. Ricketts reports two of his own cases of cirrhosis in which the same operation was performed.

11. Epiplopexy or omentopexy, variously described as the Talma, the Talma-Drummond, or the Morison operation, has been employed oftener than any procedure devised (see below).

12. Eck's fistula, which is made by establishing a communication between the portal vein and the vena cava. Vidal's (1903) operation of this nature has already been discussed.

13. Splenopexy, or anchoring the spleen to the anterior peritoneal wall, with or without omentopexy, has given some very favorable results.

14. Multiple visceropexy is suggested by Ricketts as a procedure that might accomplish much good.

15. Splenectomy has been employed by Mayo (1918) and others, on the theory that the hepatic cirrhosis might be secondary to metabolic poisons derived from a diseased spleen. Mayo suggests, however, that removal of the spleen diverts all the blood from the general circulation which would normally reach the liver by way of the splenic vein, and thus relieves the subnormal liver of an overload. And he reports that of five patients with portal cirrhosis treated by splenectomy four recovered and were markedly improved. But the query propounded by J. Bapst Blake (1918), when this subject was under discussion by the American Surgical Association, remains unanswered: *Does* removal of the spleen diminish the *hepatic* circulation?

We believe that in seeking operative relief for the symptoms of portal cirrhosis, the surgeon always should bear in mind what the two chief symptoms are—ascites and hemorrhages; and that remembering the probable pathogenesis of each of these symptoms he should adopt his plan of operation accordingly. If the hemorrhages are the predominant feature, measures for establishing a collateral circula-

tion are indicated; if there are no hemorrhages, but ascites is annoying, it is probable that complete evacuation of the fluid by laparotomy, with such alteration in the nutrition of the serous surfaces of the peritoneum as accompanies this simple operation, will be as effective in relieving the ascites as will any more complicated procedure. We quite agree with Bogojawlensky (1909) who claimed that the benefit of the operation of omentopexy is due to the laparotomy and consequent hyperemia rather than to the fixation of the omentum, although the latter may help. He induces as much hyperemia as possible during the operation. Bogojawlensky also claims that it is essential that all ascitic fluid be removed from the peritoneal cavity on account of the danger of too rapid absorption if the kidneys are not functionating properly. Dock (1910) also is of the opinion that the relief of the ascites may be due more to the operation on the serous membrane than to the opening of collateral circulation.

The most efficient method for the **establishment of a collateral circulation** is **epiplopexy** (omentopexy). Talma began his studies on this subject in 1889; but the first case in which the operation was successful was published by Drummond and Morison in 1896. We do not believe that this operation is indicated in cases of cirrhosis unaccompained by gastro-intestinal hemorrhages; and when its performance has been followed by the disappearance of an uncomplicated ascites, it is highly probable, as noted above, the success was attributable to other factors in the operation and not to the epiplopexy itself.

Complication of cirrhosis with nephritis does not necessarily contraindicate Talma's operation; but any operation is contraindicated when the patient is markedly weakened by disease of the heart and kidneys; and is absolutely contraindicated when the functional activity of the liver cells has been abolished, as shown by the presence of urobilinuria and acholia. Long-continued jaundice acts as a contraindication on account of the predisposition in these cases to postoperative hemorrhage.

The results of the operation of epiplopexy, undertaken for the relief of ascites in cirrhosis of the liver may be seen from the figures collected by Ricketts: he notes 1565 cases in which the operation of epiplopexy was performed, the results being as follows:

Patients cured	30.4 per cent.
Patients relieved	19.8 per cent.
Patients unrelieved	39.2 per cent.
Patients died	10.6 per cent.

McWilliams (1907) quoted the following statistics:

Author	Cases	Symptomatic Relief
Koslowsky	168	46 per cent.
Greenough	105	42 per cent.
Monprofit	224	35 per cent.

Bunge reports 33 per cent. of permanent cures and 33 per cent. improved.

The report of Dock illustrates the usual course of those cases which are ultimately cured of the ascites. In this case tapping was resorted to fourteen times and was followed by omentopexy, performed in 1902 by Edward Hamilton of Houston, Texas; during the following seven months paracentesis was performed ten times for the relief of the ascites. These tappings were followed by permanent relief, internal medication fully controlling any slight subsequent recurrence of the ascitic fluid during the seven years which had elapsed since operation up to the time of Dock's report in 1909.

The *technique of the operation* is discussed in Chapter XXIV.

For the **relief of the ascites,** as already pointed out, it is more rational to resort to laparotomy, with sponging of the parietal peritoneum and of that covering the liver; or even a resort to hepatopexy as practised by Delagenière. The performance of epiplopexy at the same time is not to be condemned, since it may be of benefit in relieving unrecognized gastro-intestinal varices.

Finally **removal of the spleen** must now be given due consideration in cases of portal as well as in those of biliary cirrhosis; for even though the *modus operandi* of this method of treatment remains in doubt, there can be no doubt that in selected cases it has proved its value.

Cysts of the Liver

The most common cyst of the liver is the **hydatid** or **echinococcus.** Though knowledge of hepatic cysts dates back to the times of Hippocrates and Galen, and though the anatomists of the sixteenth and seventeenth centuries accurately described such cysts, in those times the cysts were supposed to be enlargements of the lymphatics. Pallas (1760) proved that the cysts were independent parasites, and also showed the close relation they held to the tape-worm. Bremser, in 1819, published the first accurate account of the echinococcus occurring in the human liver. The subject was thoroughly discussed by Davaine in his treatise on intestinal parasites, first published in 1860.

The exciting cause of the echinococcus cyst is the *Tænia echinococcus* (*Echinococcus granulosus*), a parasite found in the upper intestinal tract of several animals, such as the dog, the wolf, and the jackal. Richardson, in 1867, and later Madelung (1885) have shown that the echinococcus is also found in sheep. Kehr (1904) states that the domestic cat and rabbit may also be the source of infection.

The ova enter the gastro-intestinal tract of man with food or drink, or possibly as the result of handling or being licked by an animal infested by the parasite. The capsule is digested in the intestinal tract, and the embryo is liberated. In the larval state the echinococcus is globular in form. It possesses six hooklets and four suction discs which aid it in boring into the tissues. It finds permanent lodgment in various portions of the body; in the liver, spleen, kidney, lungs, etc., the liver being the most frequent site of its lodgment and development. It is probable that the larvæ enter the radicles of the portal vein and are then carried directly to the liver. Some may also reach the liver through the bile-ducts. Douglas (1909) quotes Davaine as having found the echinococcus in the liver in 166 of 376 cases; Böcker, in 27 of 40 cases; and Weisser in 451 of 900 cases. The ova as a rule do not escape again from the human body, though they are discharged constantly from the bodies of sheep in the slaughter house, to be devoured by dogs and again developed into tape-worm (Morris). The proper means of *prophylaxis* of hydatid disease are thus indicated, in guarding the food supply of dogs, and in proper attention to their dejecta.

Age and Sex.—Echinococcus disease is seen most frequently in those between the ages of twenty and thirty years. It may be found at any age; even in the fetus the cyst has been found of such size as to prevent delivery. It has no predilection for either sex.

Distribution.—Echinococcus disease is found most frequently in Iceland. In that country the close association of mankind with dogs is marked, and this fact is held accountable for its frequency. According to Morris (1888) one-seventh of the human mortality in Iceland was due to hydatids. The multi-locular cyst rarely is seen in Iceland. In Australia and Italy the disease is not uncommon. France and Germany are rather free from it, while in England and the United States it is comparatively rare.

Pathology.—After the parasite (in larval state) reaches the liver it loses its hooklets and enters the immature or cysticercus stage. Inflammatory changes cause a protective connective-tissue encapsula-

tion. The cyst-wall consists of *two layers*, an outer laminated membrane or capsule; and an inner vascular layer variously designated as the parenchymatous, granular, or germinal layer. The contents consist of a clear, colorless, transparent fluid, non-albuminous, of a specific gravity of from 1000 to 1015. It contains sodium chloride and traces of succinic acid and of sugar. When the capsule is broken down either by erosion or suppuration, or when the cyst becomes infected with pyogenic bacteria, the fluid becomes turbid; and sometimes it is bloody or bile-stained. Boinet in studying hydatid fluid extracted a ptomain from it in the form of a prismatic crystal, fern-leaf in shape. The death of a mouse occurred five minutes after the administration of three-sixty-fourths of a grain under the skin. A larger dose given to a rabbit caused convulsions, alteration in the rhythm and rapidity of the respirations, tachycardia, dilated pupils and collapse, the symptoms usually assigned to hydatid intoxication. Fowler states that this toxin is much more abundant in cases in which puncture and electrolysis have transformed the clear fluid into a turbid syrupy fluid which is rich in albuminoid matter.

When the cyst is fertile, daughter cysts develop within the original or parent, cyst; other, or granddaughter cysts, at times develop within the daughter cysts. The heads or scolices of the parasites are found on the inner surface of the germinal layer, in pedunculated vesicles called "Brood-capsules." The walls of these vesicles are similar to those of the primary cyst. A single scolex or several scolices may be found in each of the brood-capsules. They are like the parent parasite, having the same number of hooklets and suction discs. The scolex may be free in the capsule; if the capsule ruptures, the scolex will then be found free in the cyst. In some instances deposits of lime salts will be found in the scolex.

After the cysts have undergone degenerative changes, the hooklets and portions of the cyst membrane may be found in the resulting detritus. Where calcification has taken place, it often will be difficult to recognize or determine the causes or origin of the cyst.

Prudden (1911) cites two rare forms of echinococcus cysts, in one of which, *echinococcus scoleciparicns*, the secondary vesicles are formed on the outside of the primary cyst-wall. The second variety, *echinococcus multilocularis*, which is more common in man than the former, results from disturbances in the development of the cysts. In an encapsulated mass, series of irregular cysts will be surrounded by bands of connective tissue of varying widths. This has been called by Vierordt "echinococcus alveolaris." A cross-section of the tumor

gives an appearance which accounts for the term "alveolar colloid" which was formerly applied to it.

Thompson (1896) exhibited an interesting specimen of multiple hydatids. "The liver was the seat of extensive growths, there being five distinct tumors in various stages of activity. Two had suppurated, two had died and had been converted into inert masses, while the fifth was in the active growing stage." Multiple cysts occur in about 12.5 per cent. of cases. Their existence may be suspected at operation if evacuation of the first cyst does not cause a sufficient diminution in the size of the liver.

Echinococcus cysts of the liver generally form in the right lobe, and in about 90 per cent. of the cases the cyst is solitary. Any part of the liver may be involved. The size and shape of the organ vary with the position of the cyst. If it is in the centre of the liver there generally is a more or less uniform enlargement; if near the border or on the lower surface of the liver, or if there are multiple cysts, the shape of the liver is greatly altered. Cysts on the lower surface generally grow downward and ultimately may fill the greater part of the abdomen, even reaching the pelvis, as in a case reported by R. S. Fowler (1906). When growing on the upper surface of the liver, the pressure against the diaphragm may be great enough to compress the overlying lung. Usually a tumor is noticed in the right hypochondrium or in the epigastrium.

Echinococcus disease may last for years. The growth of the cyst is slow, the course of the disease extending over a period of from two to thirty years. It may exist for years without giving rise to symptoms and may be discovered or suspected only after the abdomen has been opened at autopsy. In an analysis of twenty cases, Barrier, (1840) found that in three the disease had lasted two years; in eight, from two to four years; in four, from four to six years; and in the remaining fifteen cases the duration was fifteen, eighteen, twenty and even thirty years. In fourteen cases studied by Henry Morris (1888), the average duration of symptoms was about seven years. In the series of cases studied by Cauchoix (1908), the probable duration of the disease before treatment was sought is indicated in fifteen instances: this period varied from a few days to thirty-six years (Obs. xxxvi), the average duration of symptoms being more than five years.

The *untreated hydatid* may progress indefinitely, death from another cause intervening. Death may result at any time from rupture as a result of trauma, or from infection with suppuration; or spontaneous rupture without infection may occur. In cases where the parasite

dies, or ceases to grow, the cyst usually atrophies, and the contents of the sac resemble sebaceous matter, or are greatly altered by calcareous changes. A *spontaneous cure*, of course, results. If the cyst opens into the *biliary apparatus*, sterile bile as a rule will kill the parasite and effect a cure. If the bile is infected, however, suppuration of the cyst will ensue. *The cyst may rupture spontaneously* into one of the hollow viscera, such as the stomach or intestine, into the lung or pleural cavity, or into the free peritoneal cavity. When the stomach is invaded the contents of the cyst will be vomited; if the lung is the organ involved violent coughing will cause the expulsion of the fluid, and sometimes of unbroken daughter cysts; if rupture has taken place into the intestine, the contents of the cyst may be recovered in the stools. When rupture takes place into the general peritoneal cavity, the result will depend upon the condition of the cyst contents: if there is infection present, a suppurative peritonitis will follow with a mortality of about 90 per cent.; if the contents are sterile, multiple cysts may form in the the peritoneum, unless the patient succumbs to the septic condition caused by the absorption of the ptomains and toxins contained in the fluid. A diagnostic point of some value in these cases is the urticarial rash which develops rather rapidly. Rupture of the cyst may be external in rare cases; occasionally it ruptures both internally and externally. Dévé (1907) describes **hydatid gaseous cysts** (*Pneumokystes hydatiques*) of the liver, a condition first mentioned by Laennec; the condition is attributed to rupture of the echinococcus cyst into the stomach, intestine or lung; rarely it is caused by putrefactive changes within an unruptured cyst. The term is also applied to an hydatid cyst to which air has been admitted as the result of operative procedures, thus being analogous to the term "open pneumothorax." Dévé collected fifty-one cases of hydatid pneumocysts; operation was done in fourteen cases with eight deaths resulting.

Symptoms.—Echinococcus disease may extend over years without giving rise to symptoms. Unless there is infection of the cyst, the symptoms, as a rule, will be limited to the effects of the pressure exerted by the tumor. At times the patient may complain of a dull, dragging feeling, with some discomfort and slight pain in the epigastrium, loin, or back. When the cyst is large and situated on the upper surface of the liver, pressure on the diaphragm may cause interference with respiration. In such cases the liver will be pushed downward. When the cyst grows large enough to interfere with the normal functions of other organs, pressure symptoms referable to the involved organs will be noted. Pressure on the biliary ducts may cause jaundice;

pressure on the inferior vena cava will cause ascites or edema of the lower extremities; pressure on the portal vein will cause gastro-intestinal varices or ascites; pressure on the gastro-intestinal tract will cause functional disturbances. Attacks of urticaria are not uncommon.

When large enough to be palpable, the connection of the tumor with the liver is evident; its surface is smooth and rounded, imparting to the examining fingers the sensation of a dense fluctuating mass. When the tension within the cyst is high, fluctuation is not present and the tumor seems a solid mass. A jelly-like feel is absent in the great majority of cases; Finsen was unable to detect it in a series of 235 cases. If adhesions are present, the tumor is more or less fixed; if absent, the tumor moves with respiration. There is no tenderness on pressure, as a rule, unless the cyst is infected.

Hydatid fremitus, a tremulous impulse felt in palpation, over a hydatid cyst, may or may not be elicited. It is supposedly caused by the impact of the daughter-cysts against one another. If, however, the parent sac contains fluid the daughter-cysts will be suspended and will not give this sign.

A sign that is considered pathognomonic of hydatid disease by Santoni was described by him in 1894, when he found that auscultatory percussion revealed a peculiar sound, or booming, of low tone, lasting but a short time and ceasing abruptly. The mass is dull on ordinary percussion.

After *infection of an echinococcus cyst* has taken place, the symptoms presented are much more intense and generally alarming. The patient becomes septic, with irregular temperature, abdominal pain and marked tenderness over the region of the tumor. Symptoms of peritoneal irritation usually are marked.

The symptoms presented in echinococcus disease are often very misleading, as may be seen from the following case history:

L. E., female, aged twenty-two. Born in Russia; hat-trimmer. Admitted to the German Hospital, December 14, 1909. Family history negative. No tuberculosis. No carcinoma. Has been in this country five years. Well until present illness. No acute diseases, no operations. Habitual constipation. Menstrual history surgically negative.

Present Illness.—Began to "feel badly" two days before admission, complaining of indigestion which she had had at irregular intervals during past three months. Her attacks of indigestion began about two hours after meals, and were characterized by a sensation of discomfort and bloating in abdomen. No nausea or vomiting. Present attack began with pain in epigastrium, a little to right of median line, midway between costal margin and umbilicus. This has remained

the most acutely painful point, but pressure over any portion of the abdomen caused pain. Patient had never been jaundiced.

Physical Examination.—Well-developed and well-nourished girl, expression worried. Face flushed, cheeks hectic. Knees kept flexed. Respiration costal. Head negative. Tongue slightly coated. Chest and heart negative. Abdomen full but not distended. Abdomen generally rigid and in upper portion hard. Tender everywhere. Liver dullness completely obliterated. No mass palpable. No marked dullness in flanks. Peristalsis present. Pelvis normal with exception of tenderness on pressure throughout vaginal vault. Pulse rapid but regular, volume small, tension high.

Hb. 74 per cent.; leukocytes, 24,550; polynuclears, 88.5 per cent.

Provisional diagnosis of duodenal ulcer with perforation was made, and operation advised.

Operation on day of admission. Ether anesthesia. Upper right rectus incision. Intestines distended. Stomach, gall-bladder, duodenum and pancreas apparently normal to palpation and inspection. Palpation of liver revealed a nodule situated in the left lobe on upper surface, anteriorly. Area of yellow-white tissue protruded from surface, about the size of top of wine-glass. Incision made into tumor, through very tough wall. Cavity about size of lemon revealed. Cavity filled with echinococcus daughter-cysts, not infected. Cavity thoroughly curetted and packed with gauze. Wound closed around drainage.

December 27, 1909: Course of convalescence normal. All gauze removed to-day. Cavity in liver granulating.

December 28, 1909: Patient up and around ward, talking with other patients. Complained of sudden sharp pain over heart, with sudden cessation of heart action and of respiration. Was dead within four minutes.

Autopsy revealed a second cyst about the size of a lemon situated in the right lobe of the liver. No evidence of peritonitis. No demonstrable cause of death.

Diagnosis.—It is practically impossible to recognize hydatid cysts of the liver, when the tumor is small. When the cyst is palpable, its evident connection with the liver, and its generally rounded, smooth surface are suggestive features. Unless fastened in place by adhesions, it moves with respiration. A clear history of the course of the disease from the first symptom presented usually helps to clear up the differential diagnosis. When the cyst has become infected the diagnosis is comparatively easy, especially in those cases where echinococcus disease is known to exist. There will be marked pain, an irregular temperature, and symptoms of peritoneal irritation in addition to those formerly presented by the disease. These symptoms may be less marked, as pointed out by Kehr, when the tumor becomes adherent to the abdominal wall and threatens to break through the skin. The diagnosis must then be made from the redness and the peculiar condition of the skin and subcutaneous tissues, together with the general symptoms presented.

In reaching a diagnosis of hydatid cyst, the usual causes of tumors of the liver must be considered and excluded. Carcinoma, abscess, syphilis and tuberculosis are the underlying causes of most liver tumors. Sarcoma, lymphadenoma, angeioma, myxoma, fibroma, and atheroma are also observed, but not so frequently. Tumors of the adjacent organs must also be considered. Those most likely to throw doubt on the diagnosis are tumors of the gall-bladder, of either kidney, or of the spleen. Empyema and subphrenic abscess may also be confounded with hydatid cyst.

Primary carcinoma (page 579) of the liver is rare. Usually carcinoma is secondary to a similar growth in some other organ; the presence of carcinoma in a locality other than the liver makes the diagnosis of carcinoma of the liver, when a tumor of that organ is discovered, most probable. The carcinomatous tumor is more irregular than the cyst, presents a roughened surface, and feels harder than the cyst. There is increasing pain in the region of the liver, with decrease in weight, loss of appetite and of strength. Emaciation soon becomes marked, while the liver shows increase in size, and becomes tender to the touch. Jaundice may be early or late in the disease and always increases in intensity.

In *abscess of the liver* (page 540) a palpable tumor may develop either in the epigastrium or beneath the right costal margin. It is tender on palpation, usually increases rapidly in size, and gives marked constitutional symptoms, such as chills, sweating, irregular temperature, and a peculiar sallowness of the face. The previous history usually shows an antecedent focus of pus in some other region of the body, especially in the appendix, or the occurrence of dysentery. The stools should be carefully examined for amebæ; a blood examination usually shows a marked increase in the white cells. This is not the case in an uninfected hydatid cyst. The differential diagnosis between an infected suppurating hydatid cyst, and an abscess of the liver often is extremely difficult, and depends more upon the clinical history than upon the physical examination.

Syphilitic tumors (page 576) of the liver are much more common in this country than hydatid cysts. In the cases of recent gummata, there usually is a distinct tumor, either in the form of a hard nodule or a large flat mass. Usually a clear history of specific infection can be obtained. In every instance where syphilis of the liver is suspected, potassium iodide should be administered. This will cause some diminution in the size of a syphilitic tumor, although it may not entirely disappear. The Wassermann test also should be made.

Tuberculosis of the liver at times may cause a distinct tumor, as shown in the case reported by MacKenzie. In this case there were multiple abscesses of the liver, with a globular swelling about the size of an orange in the right lobe. In such instances, the symptoms presented are similar to those of chronic abscess of the liver, with acute exacerbations.

The other tumors of the liver seldom are seen and do not present symptoms which would allow a differentiation from the noninfected small hydatid.

In *empyema* and *subphrenic abscess*, there usually is a history of pneumonia, pleurisy, cholelithiasis, cholecystitis, appendicitis, or some other lesion which leads to a correct differentiation between the condition present and an echinococcus cyst.

Ghedini (1907) claims that the presence of an echinococcus cyst in the body may be revealed by the *hemolytic blood test.*

Treatment.—Other things being equal surgical treatment is indicated in every case of hydatid cyst of the liver as soon as the diagnosis is made. There is nothing to be gained by waiting, and medicaments are totally powerless to destroy the worm. It will be convenient to study first the treatment of the simple, noninfected, hydatid cyst of the liver; and then to discuss the proper treatment of the various complications that may arise (suppuration, rupture, etc.). The methods for prophylaxis have already been mentioned (page 560).

Method of access for evacuation of an echinococcus cyst deserves a few words. Most cysts develop downward and are best exposed by laparotomy; resection of the costal border or division of the suspensory ligament may be necessary for better exposure. For those growing upward beneath the diaphragm the operation is similar to that for the drainage of an abscess of the liver by the transpleural route (Israel, 1879). Roser claims that he proposed this plan as early as 1864.

Treatment of Single Uncomplicated Cysts.—*Puncture or aspiration* of the contents of the cyst was the earliest form of surgical treatment adopted. The fact that cures were reported from these simple methods merely shows that the patients were not kept under observation long enough after operation to exclude the possibility of recurrence. This may not take place for many months, or even years. If infection occurred, either before or after inspiration, it was recommended to open the cyst widely and drain it; under such circumstances it usually was adherent to the abdominal wall. The

method of opening and drainage appears first to have been erected into a principle, applicable to all echinococcus cysts of the liver, by Landau in 1880; the procedure is termed marsupialization of the cyst, that is, converting it into a pouch.

Marsupialization.—The cyst is exposed by laparatomy, its walls are sutured to the edges of the abdominal incision, and it is opened then or at a subsequent operation. Its contents are evacuated, including detached daughter-cysts, and such scolices as have escaped from their brood-capsules; the inner capsule of the cyst (germinal layer) is also removed. The outer capsule should not be disturbed; it is closely surrounded in most cases by dilated biliary channels (perhaps containing infected bile) and by blood-vessels (especially portal or suprahepatic veins) of unknown size which may give rise to troublesome or even fatal hemorrhage. The cyst having been thus emptied, its cavity is stuffed with gauze, and allowed to heal by granulation. Though the immediate mortality of this operation is low, Cauchoix (1908) noting ten deaths among 185 operations recorded by Vegas and Cranwell (1901), it has manifest disadvantages in the form of postoperative complications, which no longer commend it to surgeons. Chief of these objections is the long period of convalescence: Cauchoix found that though the great majority of cysts were completely closed in from one to four months, yet that in many patients the fistula continued to discharge for six months or a year. Moreover, it is very difficult to prevent secondary infection when marsupialization is employed, the case being analogous to that of a cold abscess opened and drained; and suppuration in an opened hydatid cyst may become a very serious matter. The secondary discharge of bile which often occurs through the fistulous tract may seriously impair the patient's health. These biliary discharges as well as spontaneous hemorrhages into the opened cyst, are due to the negative pressure created in the cyst by opening and drainage.

Suture of the incision in the cyst, after the evacuation, and closure of the abdominal wound, was a method introduced in 1883 by Knowsley Thornton and popularized in 1891 by Bond. The sutured incision in the cyst-wall was fixed to the abdominal wound. In this way it was hoped to avoid the disadvantages attendant upon prolonged drainage, at the same time permitting secondary opening and drainage of the cyst should occasion demand.

Reduction of the evacuated cyst, without suture of the incision in its walls, and closure of the abdominal wound without drainage, was employed by Ryan and by Hamilton Russell in 1894 and adopted by a

few other surgeons; but the dangers of secondary infection of the peritoneum through bile or blood effused into the cavity of the unsutured cyst soon caused this method to be abandoned.

Endocystorrhaphy (*Capitonnage*) was adopted in 1896 by Delbet and by Posadas. They diminished but did not entirely obliterate the cavity of the cyst, after its evacuation, by interrupted sutures, cautiously passed through its walls. The cyst was completely closed, and was fixed to the abdominal wound, which was sutured without drainage.

Enucleation of the cyst from the surrounding hepatic tissue has been employed in a few cases; but the danger of opening into blood or bile-channels renders it a most unsuitable method. Cauchoix refers to fourteen cases, in four of which the operation could not be completed.

Extirpation of the cyst may be done when it is pedunculated; or a small portion situated within the liver may be removed by partial hepatectomy. Fowler prefers this to any other method, whenever practicable. Its advantages are a shortened convalescence, freedom from a biliary fistula, and assurrance that the entire disease has been removed.

Prophylactic Treatment (*Formolization*).—Under this title, Cauchoix describes a method of sterilization of the cyst contents by injection of a 1 per cent. formalin solution with the view of preventing recurrence of the disease. It was demonstrated experimentally by Dévé (Thèse de Paris, 1901) that each of the parasitic elements contained in echinococcus cysts is capable of reproducing the primary lesion; and Cauchoix showed that such recurrence occurred clinically, though often not for years after the primary operation. After various other antiseptics had been tried with no very marked success, Quénu (1902) adopted formalin solution (1 per cent.) as the sterilizing fluid, and employed the following technique in a number of cases with marked success: The cyst was exposed by laparotomy, and thoroughly isolated by gauze packing. The fluid contents of the cyst were then withdrawn through a trocar and canula by syphonage into a funnel. To avoid possibility of soiling the surrounding tissues, a very fine canula was employed and the trocar was passed through the wall of the rubber tubing attached to the end of the canula, so that when it was withdrawn no leakage occurred in its tract (Fig. 154). When

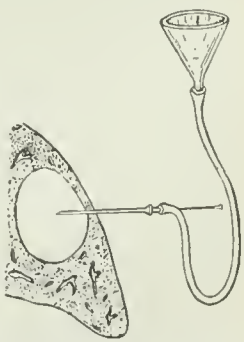


Fig. 154.—Quénu's Method of Formolization of Hydatid Cysts (*Ashhurst*).

the cyst has been thus evacuated, the funnel is emptied, and is then filled with the formalin solution which is allowed to enter and distend the cyst, by the force of gravity. This solution is left in the cyst for five minutes, and is then withdrawn. The cyst, thus sterilized, is incised freely, and the germinal membrane is removed. Repeated laboratory examinations of this membrane showed that all parasitic elements had been killed by the formalin solution. The cyst therefore may be closed with impunity, restored to the abdomen and the external wound closed without drainage. It always is well, however, to fix the cyst-wall to the abdominal wound so that should an intracystic effusion of bile or blood demand evacuation this can be accomplished without wide opening of the peritoneal cavity.

This *prophylactic treatment of Quénu* undoubtedly is the best method of operation in all cases to which it is applicable; but there are a few to which it is not suited: cases, for example, when the hydatid cyst is full of daughter-cysts and contains no fluid, so that it cannot be evacuated by puncture. In such circumstances Cauchoix recommends that the surrounding tissues be protected by gauze soaked in formalin, so that the parasitic elements unavoidably discharged over the field of operation may be promptly killed.

Treatment of Complicated Cysts.—This subject has been well studied by Cauchoix (1908) and we have made free use of his valuable memoir in what follows.

Suppurating hydatid cysts require the same treatment as abscesses of the liver; but when the infection is of very low grade, as manifested by the clinical history, it may be possible to close the cyst completely after the use of a formalin injection, or at least to suture the incision tightly around a drainage tube which may be removed in about four days. According to Végas and Cranwell the *mortality* in cases of suppurating cysts treated by marsupialization is from 20 to 30 per cent.

Rupture of a cyst into the peritoneum requires immediate laparotomy and drainage, both of the cyst and of the pelvis. This is true not only in the case of rupture of a suppurating cyst, but also when an aseptc cyst ruptures, because in the latter case the secondary development of peritonitis from effusion of bile (choleperitonitis) is much to be feared.

Rupture of a cyst into the intestine is dangerous because of the secondary infection of the cyst which is nearly sure to occur. The proper treatment is laparotomy so soon as the immediate shock of the accident subsides, with repair of the intestinal defect and drainage of the cyst. Rupture occurs oftenest into the duodenum, transverse colon or stomach.

Compression of the biliary ducts by an echinococcus cyst leads soon to a condition of angeiocholitis, which demands choledochotomy and hepaticus drainage in addition to the treatment appropriate to the cyst itself.

Rupture of a cyst into the biliary passages is by no means rare. It was carefully studied by Quénu and Duval in 1906. In most cases this occurrence is attended by severe pain, and the appearance of septic symptoms, chills and fever. Unless the existence of a hydatid cyst is already known, the recognition of such a complication would be very difficult. The common duct may be obstructed by the impaction of some of the cyst contents, or merely by inflammatory swelling. Usually choledochotomy and hepaticus drainage will be required, in addition to proper treatment of the cyst. In six operations in which cholecystostomy was done, with drainage of the cyst, there were two deaths. In eight operations on the common duct there were three deaths (Quénu).

Rupture of a cyst into the thoracic cavity is another very fatal complication. Even if it ruptures only into the *pleura*, symptoms of pulmonary distress are usual; the pleural effusion is blie stained (cholethorax), owing to leakage of bile into the cyst cavity. Cauchoix collected five cases, with three deaths. The proper treatment is pleurotomy, with free drainage, and marsupialization of the cyst. If rupture into the *lung* or *bronchial tubes* occurs, death is almost inevitable. Treatment is the same as for pulmonary abscess; the hepatic cyst should be drained also, preferably through the thoracic wound.

The mildly infectious nature of some suppurating hydatid cysts is exemplified in the following case, in which the cyst was opened and drained without infecting the general peritoneal cavity.

M. B., male, aged twenty-two, born in Austria-Hungary, admitted to the German Hospital (Medical Department), October 15, 1909. Came to the United States three years ago, since which time he has been employed as a butcher. Denies venereal infection. Family history negative. Had attacks similar to present, eight years ago. Has had no other illness of any kind.

Present illness began two weeks before admission. Had sudden attack of severe abdominal pain, greater in epigastrium and radiating across upper abdomen and into back. Pain severe and lasted several hours. There was some epigastric swelling. Vomited after eating. Took castor oil and felt better. Had second attack five days before admission, with vomiting. Third attack began day before admission, and lasted two hours. Vomited after drinking glass of milk. During attack of pain felt hot and cold alternately and had a slight chill. Appetite remained good. Bowels fairly regular until lately.

Physical Examination.—Fairly well-nourished man, face covered with acne. Tongue heavily coated in middle and back. Teeth in fair condition. Head, lungs and heart negative. Abdomen flat. Liver extends from fifth interspace to 6 cm. below costal margin. Edge rounded. In epigastrium and slightly to left is a mass with rounded edges, firm in consistence, tender on pressure. Moves with respiration. Spleen not palpable. No rose spots. Extremities negative.

October 16, 1909: Yellowish-brown, liquid stool. Occult blood absent. Trace of bile. Alkaline reaction. Soaps, neutral fats and fatty acid crystals. Many yeast cells. Few epithelial cells.

Stomach test-meal: Semi-liquid stomach contents, not completely digested. Acid in reaction. Free hydrochloric acid, sixty-four. Total acidity, ninety. Occult blood absent. Bile absent. Starch, neutral fats, epithelium, yeast cells present.

X-ray examination showed shadow to right of median line extending from right costal margin half way to umbilicus, affected slightly by deep inspiration. Continuous with liver shadow.

October 20, 1909: Hemoglobin, 95 per cent.; leukocytes, 11,200.

October 26, 1909: Temperature, 99°; respiration, 24; pulse, 84.

October 27, 1909: Temperature, 103°; respirations, 26; pulse, 104. Patient seized with attack similar to those described. Severe pain in upper abdomen radiating to back followed by rise of temperature and pulse rate. Swelling in epigastrium very tender. Has persisted and extended more to right. Leukocytes, 16,500. Polynuclears, 84 per cent.; lymphocytes, 8.5 per cent.; mononuclears, 1.5 per cent.; eosinophiles, 2 per cent.; basophiles, 0 per cent.; transitional, 3.5 per cent.; unidentified, 0.5 per cent.

Transferred to surgical ward. Patient lies on side with head down and knees drawn up. Swelling in epigastrium visible, about size of an orange, exceedingly tender, dull on percussion, evidently continuous with liver. Moves very slightly with respiration.

Operation by Dr. Deaver, October 30, 1909: Ether anesthesia. Upper right rectus incision. Left lobe of liver found lower than usual, the convexity being boggy. Few adhesions between left lobe and surrounding structures. In liberating the adhesions, wall of a purulent collection in the left lobe was ruptured and thin stream of pus was liberated. Attempts at aspiration were futile. Surrounding region well protected by means of gauze pads, mass incised and large quantity of pinkish-yellow faintly odorous fluid liberated. The fluid contained many white cyst-like bodies in a state of collapse, resembling white grape skins, varying in size from a small pea to a bird's egg. About a pint of material removed. A cavity about the size of a large fist remained. This was curetted carefully, and then drained with large rubber tube and gauze packing. Wound closed to edge of drainage.

November 12, 1909: All gauze removed under primary chloroform anesthesia. Gauze packing introduced.

November 22, 1909: Gauze removed, followed by cyst-wall.

November 26, 1909: Cavity granulating. Wound discharging considerable bile.

Discharged from hospital December 24, 1909, with small sinus. No discharge of bile.

Temperature ran an irregular course, until the nineteenth day after operation, when it reached normal and remained so.

Non-parasitic cysts of the liver may be divided into two classes, the *congenital* and the *acquired*. Among the former are dermoid cysts and multiple epithelial cysts. The former are operable. The latter are of interest mainly from a diagnostic point of view, as they are inoperable and a fatal result usually occurs from the "cystic degeneration" of the liver, which is frequently found associated with cystic degeneration of other organs such as the kidneys, ovaries, etc. Death usually results from obstruction of the portal vein. When such cystic disease of the liver is discovered at operation, further operative procedure is contraindicated.

Kehr quotes Pellman as dividing into five classes the non-parasitic cysts of the liver which may be treated by operation:

1. Retention cysts of the biliary tract.
2. Cysts lined with ciliated epithelium, said by von Recklinghausen to be due to retention of mucus.
3. Dermoid cysts.
4. Epithelial cysts, or cystadenomata.
5. Lymph cysts.

The symptoms produced by any of these various classes of cysts are similar to those of the non-infected echinococcus cyst (page 563). These cysts may attain considerable size, and then may be mistaken for cystic conditions of the kidney or ovary. The differental diagnosis is to be made from study of the symptoms presented, together with the anatomical diagnosis of the organ involved. Non-parasitic cysts of the liver cannot be differentiated before operation from echinococcus cysts of the liver, and often it is necessary to depend upon the microscopical examination of the contents or of a section of the cyst-wall for a correct diagnosis. It is not justifiable in any instance to make an exploratory puncture for the purpose of diagnosis.

Treatment.—The treatment consists in the removal of the cyst contents and of as much of the cyst-wall as is possible. In pedunculated cysts, the entire mass usually may be removed. When the cysts are deep seated, an incision through the liver substance must be made, down to the cyst-wall, when it will be found feasible to shell out many of them, without rupture, owing to the fact that the cyst-wall usually is dense. When rupture during removal does occur, there is less danger of serious infection than from a ruptured echinococcus cyst. The postoperative course and treatment are similar to those following the operation for echinococcus cyst. Boyd (1913) collected 34 operations for non-parasitic cysts of the liver, with 11 deaths.

CHAPTER XIX

TUMORS OF LIVER, GALL-BLADDER AND BILE-DUCTS

Tumors of the liver, apart from those of the gall-bladder and bile-ducts, have little practical interest for the surgeon. They are important, however, from the aspect of diagnosis; but only in a few exceptional cases, so far, has it been possible for the surgeon to attempt any radical cure by means of operation. It is possible that the future will breaden this field of hepatic surgery so that relief may be afforded a greater number of sufferers from hepatic tumors.

Benign Solid Tumors of the Liver

With the possible exception of adenoma, benign solid tumors of the liver are surgical rarities. **Angeiomata, fibromata, myomata,** etc., have been observed but are principally of pathological interest. As pointed out by Mayo (1908) a diffuse or circumscribed angeiomatous condition of the liver sometimes is found during operations on the bile-passages and troublesome hemorrhage may result if injury occurs to the dilated blood-vessels forming the tumors.

Adenoma of the liver is a comparatively rare neoplastic growth, of very obscure origin. It is not an uncommon postmorten finding, although unfrequently recognized clinically. Gordinier and Sawyer (1913) collected forty-four cases reported by various observers. Cushing and Downs (1899) found seven cases of adenomata reported among seventy-five operations for tumor of the liver. Adenomata may occur at any age, cases having been reported in patients twenty months and seventy-six years of age respectively. Of twenty-nine cases, collected by Marckwald (1896), twenty-three were in patients of the male sex.

Pathology.—Adenoma of the liver is a primary growth and two varieties are recognized: the *nodular adenomatous hyperplasia* of the liver, which is not amenable to surgical treatment; and the *circumscribed tubular adenoma* (Langenbuch). Among the forty-four cases mentioned above, twenty-eight were multiple and sixteen solitary.

In *nodular adenomatous hyperplasia* which usually is associated with cirrhosis of the liver (Simmons, 1877) there is a roughened nodular condition of the surface of this organ. Cross-sections of the liver

show pathological masses of abnormal color, not definitely encapsulated but separated from each other by fibrous changes in the parenchyma (cirrhosis). Pathologists are not agreed whether the cirrhosis causes the development of these multiple adenomata (as a compensatory hypertrophy), the view held by Rolleston (1905); or whether both pathological changes are due to the same original irritant, as maintained by Engelhardt (1898) and by Dieulafoy (1901). Brissaud (1885) followed Schuppel (1875) who held that adenoma is a stage between cirrohis and carcinoma.

The *tubular adenoma* may be single or multiple, usually the latter. The tumors usually are small, though one may reach the size of a large orange. The larger the tumor the more distinct is its encapsulation. They are grayish-white or yellow in color, unless hemorrhage has occurred into the substance of the tumor, when it has a reddish tinge, or when it has been stained green by bile. The larger the tumor the more apt is internal hemorrhage to occur. *Cystadenoma* may develop in this way or may result from degenerative changes. Metastasis is very rare but Langenbuch (1897) refers to a few reported instances.

Adenoma of the liver may be derived from (*a*) liver cells, (*b*) the intrahepatic bile-ducts, or (*c*) from adrenal rests in the liver (Rolleston). The larger tumors, and particularly those which become cystic, usually are derived from the bile-ducts. Those springing from the hepatic cells sometimes contain tubules lined with cubical epithelium, thus resembling primary carcinoma with cirrhosis. In cases of adenoma of the liver reported by Keen (1893), and by J. B. Roberts (1894), it was found that the adenomatous change was due to proliferation of coccidia.

The **symptoms** of adenomata are chiefly those of cirrhosis of the liver, the symptoms being produced by compression of the vessels and bile-ducts. In the early stages no symptoms may be produced, but as the tumor increases in size digestive disorders arise, accompanied by dull pain in the hepatic region, followed by jaundice and emaciation. Other symptoms, due to interference with the portal circulation, often develop; varicosities of the abdominal veins may be seen and ascites develops rapidly. In advanced cases a hard, circumscribed mass may be felt, at times pendulous, but moving freely with the liver during respiration. In single tumors, the liver may be of normal size, but in multiple adenoma the liver is enlarged and nodular.

The **prognosis** is bad unless extirpation is possible before the development of obstructive symptoms. But the condition may exist for a long time without giving rise to symptoms of any kind.

The **treatment** is palliative in nodular adenomatous hyperplasia. Even in the case of single adenoma, operative interference is contraindicated after the onset of jaundice and ascites. In the earlier stages, exploratory laparotomy should be done: if the tumor is in a favorable location and easily accessible, it should be removed by partial hepatectomy. The operative mortality is about 15 per cent. (page 582).

Gummata of the liver usually are discussed in connection with hepatic neoplasms; they occur as single or multiple nodules varying in size from a pea to a hen's egg. They are of interest to the surgeon from a diagnostic standpoint, operative interference seldom being adopted except in cases where a wrong diagnosis has been made, or for degeneration or calcification of the gummatous nodules. When first formed the gumma is soft; later a central area of necrosis appears, and if healing is uninterrupted, a stellate fibrous cicatrix results, which is quite characteristic and usually easily recognized. Sometimes the gumma becomes calcified. It very rarely undergoes liquefaction necrosis or suppuration from secondary infection. The larger nodules are found principally on the surface of the liver, in the vicinity of the suspensory ligament, although they may occur along the free border or along the upper surface of the right lobe. Occasionally they are pedunculated (Cumston, 1903) and may be mistaken for a linguiform lobe (page 535) or a floating liver. Between the gummata may be found cicatricial bands, which penetrate into the liver in the form of deep furrows. When these are present in great numbers a condition known as botryoid liver results, the organ being divided into lobulated masses separated by the cicatricial bundles.

The *symptoms* of gummata of the liver rarely are conspicuous. As a rule the liver is enlarged and this forms the most constant symptom. Pain may be present on account of the involvement of the serosa. At times a friction rub may be detected. In the cicatricial stages, pain is almost constant, being of a very dragging character, and referred to the hepatic region, sometimes to the right shoulder. In these cases there is almost constant discomfort, exacerbations or pain being noted. Fever is never present unless there is breaking down and ulceration of the gumma. Kirchheim (1911) reported three cases of febrile syphilitic lesions in the liver, all three involving the adjoining diaphragm; in one case there was perforation of this structure followed by empyema and chronic inflammation of the lower lobe of the right lung; pleurisy with effusion developed in the second case; in the third case although the diaphragm was involved

no thoracic symptoms developed, but the patient died from perforative peritonitis.

Jaundice is rare and occurs only in those cases where there is diffuse syphilitic involvement of the liver.

The *diagnosis* depends on the history of the patient, on the discovery of other syphilitic lesions or the evidence of past lesions; on the presence of the Wassermann reaction; and on the therapeutic test of antisyphilitic remedies. Gummata of the liver must be distinguished especially from disease of the gall-bladder, and from carcinoma. The former usually may be excluded by a careful study of the history. In carcinoma the disease does not extend over so long a time; there is no enlargement of the spleen (common in syphilis of the liver); and the patient very seldom is under forty years of age. At operation the differentiation from carcinoma may be difficult, but carcinoma of the liver almost always is secondary to a primary growth elsewhere; there never are scars of healed lesions on the surface of the liver as is frequently the case in syphilis of the liver; the carcinomatous nodules often stand forth prominently from the surface of the liver; and when cut and scraped the carcinoma gives "cancer juice" which is never the case with a gumma.

Treatment.—The usual antisyphilitic measures should be instituted. If the patient's condition is urgent, arsphenamin should be administered. In most cases the vigorous use of mercury and the iodides will bring relief of symptoms. If such treatment fails, as it usually does when the gumma is very fibrous or calcified, excision of the portion of the liver affected should be attempted. Lotheissen (1912) summarized the results of forty such operations; thirty-four patients were cured, two were improved, while four died. These forty cases do not include simple exploratory operation, with separation of adhesions, but only cases of excision.

Tuberculoma of the liver is rare. Lotheissen (1912) collected thirty-four cases, as well as thirteen cases in which there was a tuberculous abscess in or near the liver. In thirty-two instances the lesions in the liver were found only at autopsy; and in twenty-three there had been no symptoms during life to call attention to the liver. Operative treatment was undertaken in fifteen cases, but a correct diagnosis before operation was made in only three. Ten of these patients recovered.

Malignant Tumors of the Liver

Sarcoma of the liver is very rare and clinically cannot be distinguished from carcinoma. In almost all cases it is secondary to

sarcoma in some other part of the body, particularly the eye, and soft tissues of the limbs. There are a few cases on record in which sarcoma of the liver has been secondary to a primary sarcoma of bone, but in none of these was there any osseous tissue in the nodules found in the liver. Langenbuch (1897) refers to two instances of secondary chondrosarcoma of the liver. Most tumors are spindle or round celled. Lymphosarcoma, myosarcoma, and myxosarcoma also have been recorded. Melanotic sarcoma is not very rare, especially when secondary to a similar tumor of the eye (choroid).

Knott (1910) collected fifty-nine cases of primary sarcoma of the liver, and reported fifteen hitherto unpublished cases, making a total of seventy-four cases.

The *symptoms* are not characteristic. Pain, enlargement of the liver, perhaps jaundice and ascites, may develop until late in the disease. Kahlden (1897) reported a case in which the first symptom was the black color of the feces, followed by dark urine, both appearing before there was any demonstrable tumor of the liver. Fever of an intermittent type is not unusual during the course of the disease. In most cases this course is very rapid toward a fatal termination.

The *treatment* usually must consist merely in palliation of the symptoms as they develop. Early exploratory laparotomy may serve to clear up the diagnosis, and in some cases the tumors have been excised with at least temporary benefit to the patients. Knott, in his study of primary hepatic sarcoma, referred to above, found that operation had been done in twenty-seven cases with fifteen deaths, a mortality of 55.5 per cent.

In eighteen cases the tumor was excised; eight patients died, a mortality of 44.4 per cent. In six cases the tumor was inoperable; four patients died, a mortality of 66.6 per cent. In three cases the nature and result of the operation are not recorded.

Of the twelve patients who survived the operation for any length of time, the after history is known in only six cases: three of these patients died in less than four months; one had recurrence after seven months; one died at the end of nine months; and only one (Bardeleben) remained in good health for as long a period as two years.

Hemangeio-endothelio-sarcoma of the liver, of which 10 cases have been collected by Foote (1919), is a congenital disease manifesting itself within 3 months of birth by enlargement of the liver and digestive disturbances. Usually there is no jaundice, no ascites, and no metastasis, but death occurs before the sixth month from compression of the liver tissue by the growing tumor.

Carcinoma is relatively the most frequent neoplasm of the liver. It occurs in middle and advanced life[1] and is seen more frequently in men than in women. It may be either primary or secondary, the latter occurring in about 96 per cent. of the cases. Among 10,000 autopsies, Hale White (1908) found ten primary tumors and 240 secondary to carcinoma elsewhere. Secondary carcinoma is of interest from the diagnostic standpoint alone, as it is not amenable to surgical treatment. Primary carcinoma may be removed with success,[2] if the diagnosis is made early enough; but the senior author, in more than 2000 laparotomies for disease of the liver and biliary system has found no case where the primary carcinoma was at a stage where cure by radical operation might have been hoped for.

Pathology.—Carcinoma of the liver occurs in three forms:

1. *Massive carcinoma* appears as a whitish or grayish opaque mass, may grow to immense size (5-12 kg.) and usually involves the whole of a lobe, in most cases the right. The growth is well defined from the surrounding hepatic tissue, does not project markedly from the surface, and the general form of the liver is preserved in spite of the enlargement If a tumor of this kind is found in the liver it is useless to search for a primary growth elsewhere (Langenbuch, 1897), as the hepatic lesion almost surely is the primary tumor.

2. *Infiltrating or diffuse carcinoma* is very rare, and like the massive carcinoma usually is a primary growth. It gives no metastases (Langenbuch) and histologically often is mistaken for portal cirrhosis (page 553) or nodular adenomatous hyperplasia (page 574). The whole liver is affected but its form is little altered except for the increase in all dimensions. Its surface is studded with little smooth round knobs, the size of peas or cherries, and between them the liver tissue is shrunken and retracted. According to some authorities (Fetzer and Perls) this is merely the most advanced form of the massive carcinoma already described.

3. *Nodular or multiple carcinoma* is the usual secondary type and is also the most frequent primary type. Under the latter circumstances most of the nodules are regarded as metastases from one original primary hepatic tumor. The nodules, which are scattered irregularly over the surface of the organ especially at its periphery, are whitish, gray or yellowish masses from the size of a pin-head to that of an orange, though rarely larger than a walnut. They stand out from the surface of the liver; frequently cause perihepatitis, with resulting adhesions;

[1] Griffith (1918) refers to 56 cases recorded in children.

[2] Freeman's patient, operated on in 1904, was reported still in good health in 1919.

and when large often become umbilicated as the result of interstitial hemorrhages.

Beadles (1896) maintained that a clinical distinction was not difficult between primary and secondary carcinoma, even in this nodular form. *Secondary nodules* are scattered all over the liver, vary in number and size, and may leave little normal hepatic tissue visible, but they are always more or less rounded in form, and are distributed with fair uniformity, not massed particularly about the fossa of the gall-bladder or the quadrate lobe. "*Primary malignant disease* forms a hard, uniform scirrhous mass, if not involving at least close upon the superior wall of the gall-bladder." It spreads backward from this region in the tongue-like prolongations which extend beyond the general mass. This localization is characteristic, and serves to distinguish primary from secondary carcinoma, even when in cases of the former nature there are metastases from the original tumor in other parts of the liver. The main feature by which primary may be differentiated from secondary carcinoma, according to Beadles, is the apparent origin of the former from the region of the cystic duct or neck of the gall-bladder or the liver tissue immediately adjoining. Gall-stones were present in eleven out of thirteen cases of primary carcinoma of the liver; but they were found only in two of sixty-three cases of secondary carcinoma.

Primary carcinona of the liver is slightly more frequent in men than in women; but in women secondary carcinoma occurs more than twice as frequently as in men chiefly owing to the greater frequency of primary growths in the area drained by the portal vein. More than half the cases occur between the ages of forty and sixty years, but it is not an excessively rare affection even in childhood. In infants and young children, however, the disease usually is primary in the liver.

The *symptoms* of carcinoma of the liver, whether primary or secondary, are not constant. Probably the condition is diagnosed during life in not more than two-thirds of the cases. In cases of secondary carcinoma it is not unusual for the symptoms referable to the liver to overshadow those caused by the primary growth. Anorexia, gradual but progressive failure of strength and loss in weight, and vague digestive disturbances are the most characteristic symptoms; but the diagnosis must be made by exploratory laparotomy unless a palpable tumor has developed.

Cachexia, which develops early, usually advances steadily. This is especially true of the development of secondary growths in the liver, the primary growth from which metastasis has occurred very frequently not causing any cachexia at all.

Jaundice is present in about 50 per cent. of all cases. It varies in degree, especially at first, but usually increases in intensity when present and persists. It may be due to obstruction of the common duct by the pressure of a primary growth in the head of the pancreas; to secondary nodules in the lymph-nodes along the common duct; to primary foci of carcinoma in the biliary ducts; or to direct pressure on the intrahepatic ducts.

Pain may be absent in carcinoma of the liver, but there always is a general feeling of discomfort, a sense of weight and heaviness. The actual pain varies somewhat with the position and extent of the growth. When the neoplasm is deep seated there is not very much actual pain; when the capsule of the liver is involved, the pain may be sharp and cutting in character. With the formation of perihepatic adhesions, pain becomes more constant, often radiating to the epigstrium, the thorax, the back, or the shoulder, and being aggravated by motion. Colicky pain is present at times in those cases where the new growth is near the hilum; it is due to biliary obstruction.

Tenderness may be elicited on deep palpation, but it is not present in all cases.

Ascites is present in about one-half the cases. If jaundice is present the fluid usually is bile stained; otherwise it is clear. Usually it is not excessive in amount, and but little discomfort is experienced from its presence. Tapping seldom is required.

Enlargement of the liver is the most constant phenomenon, the increase in size at times being very great. The right lobe generally enlarges more than the left. The edges may be palpable, firm, and hard, but usually are irregular and more or less nodular.

The *diagnosis* often is very difficult to make, unless the cases are well advanced, or unless a primary focus is recognized. In some instances an exploratory operation may be advisable to clear up the diagnosis. The condition must be differentiated especially from abscess or echinococcus cyst of the liver, syphilis of the liver, and biliary hypertrophic cirrhosis; this usually may be done by careful study of the history and by the physical examination. At times it is impossible to differentiate between carcinoma of the liver and tumors in adjacent structures, such as the stomach, the colon, the kidney or the omentum. In these cases exploratory laparotomy is justifiable.

The *prognosis* is practically hopeless. Hale White gave the duration of life as four months after the development of symptoms in primary carcinoma and seven months after the symptoms of second-

ary carcinoma began. If a primary growth can be completely removed by excision, the chances of permanent cure are reasonably good; but there are exceedingly few cases in which this treatment is possible. Lücke's patient (1891) according to Terrier and Auvray (1898) died of recurrence two years after operation (Yeomans gives the period of survival as eight years). Freeman's (1904) was in good health fifteen years after operation; and Schräder's (1897) remained in good health seven years after operation. Hochenegg's patient survived three years, and for two years of this time was in good health (Anschütz, 1903).

The *treatment* hitherto has been considered purely palliative. This certainly still is true of secondary carcinoma where operation is contraindicated, but in the future it is more than likely that earlier exploratory operation will reveal an increasing number of cases of primary carcinoma suitable for extirpation. Operation always is contraindicated in the presence of multiple nodules, marked cholemia, advanced cachexia, and lymphatic enlargements. When the tumor is single, of primary origin, and can be readily reached, partial hepatectomy may be performed. Mayo Robson reports having exposed the liver by operation in thirty-five cases and finding three among them which he considered suitable for hepatectomy. One patient died on the table, and the other two died from recurrence in a few months. Terrier and Auvray (1898) collected thirty-eight radical operations for various neoplasms of the liver; thirty-two patients recovered, and six died from the operation, giving an *operative mortality* of 15.8 per cent. The immediate result is known in seventy-four out of seventy-five cases of partial hepatectomy for various causes tabulated by Keen (1899): eleven patients died, an operative mortality of 14.9 per cent. The immediate death rate following operation is largely independent of the nature of the growth. Yeomans (1909) collected ten operations for primary carcinoma of the liver, including a case of his own. The following list includes also an operation by Terrier, which Yeomans seems to have overlooked.

(1) Jacobs: Thermocautery and drainage; recovery; recurrence in seven months.

(2) Lapointe (Operation by Segond): Pedicle clamped and divided; death on third day.

(3) Lücke: Elastic ligature and cautery; death two years later from recurrence; (possibly a gumma).

(4) Filippini: Elastic ligature, resection two days later; recurrence and death in two months.

(5) Keen: Thermocautery of entire left lobe; recurrence and death in five months.

(6) Hochenegg: Excised, and stump fixed in abdominal wound; patient died of recurrence three years later; diagnosis doubtful.
(7) L. Freeman: Tumor isolated by gauze strips through liver substance; operation January 20, 1903; excellent health 1919.[1]
(8) Schräder: Excised, wound cauterized and fixed in abdominal wound; patient alive and well seven years after operation.
(9) Roux: Excised, bleeding controlled by suture through liver; recovery.
(10) Yeomans: Incised, curetted, packed and drained; well two months after operation.
(11) Terrier: excision; recovered, but three months after operation probably had pelvic recurrence.

Tumors of the Gall-bladder and Bile-ducts

Cystic Degeneration of the Gall-bladder.—This is a rare condition which was studied carefully by Konjetzni (1911). Licini (1911) has published another case, in which the gall-bladder was converted into a multicystic tumor the size of an average apple; there was also an adenocarcinoma of the cystic duct; and the pressure of the enlarged gall-bladder on the common duct caused jaundice to develop.

According to Aschoff and Bacmeister (1909) there is in about 3 per cent. of gall-bladders an adenomatous structure, especially at the fundus; and in cases of infection which closes these glands, cystic degeneration may occur. They believe it predisposes to the development of carcinoma.

The proper *treatment* is cholecystectomy.

Sarcoma of the Gall-bladder.—Jaffé (1920) has collected 12 cases, including one of his own, of primary sarcoma of the gall-bladder. In Jaffé's patient there had been symptoms of gall-bladder disease for two months before operation. In none of the reported cases did the patients recover permanently.

Carcinoma of the gall-bladder and bile-ducts is much more common than carcinoma of the liver. Secdarony carcinoma occurs but is of no surgical interest as the lesions rarely develop except late in a general carcinomatosis.

Primary carcinoma of the gall-bladder and bile-ducts is much more common than formerly suspected. It is found in about 5 per cent. of all cases of carcinoma. It occurs three to four times as often in the gall-bladder as in the bile-ducts. Musser, in 1889, was able to collect 100 cases, in sixty-four of which the variety of the new growth was clearly indicated. Among 3908 operations on the gall-bladder and biliary

[1] In another case (carcinoma) Freeman used strips of fascia lata, as indicated in Fig. 190 (p. 794.)

passages, performed between 1890 and 1910, W. J. Mayo found 85 cases or 2.1 per cent. of malignancy. Erdmann (1919) found 15 instances of malignancy in 224 gall-bladder operations (6.7 per cent.), and in one series of 68 operations on the gall-bladder reports encountering no less than 9 cases of carcinoma. The senior author found 2.3 per cent. of malignance in 262 operations performed by him at the German Hospital but in another series of 820 gall-bladders submitted (1916–1920) for pathological examination, only six (0.73 per cent.) were reported as being carcinomatous.

Primary carcinoma of the gall-bladder is most frequently seen between the ages of fifty and sixty years; but Pröscher reported a case in a man twenty-two years of age. It is much more frequent in women than in men, the proportion being 3–1 according to Musser (1889), and 4–1 according to Fütterer (1901). Among seven patients operated upon by the senior author, six were women varying in age from thirty-six to sixty-three; the seventh patient was a male, twenty-six years old. Another patient whose gall-bladder is represented in Plate VIII, was 70 years of age.

Schröder (1892) said that 14 per cent. of all gall-stone patients eventually suffer from carcinoma of the biliary apparatus. The theory that the irritation of the gall-stones predisposes to the development of carcinoma in the gall-bladder is well borne out by statistics: thus Musser found gall-stones associated with carcinoma in 69 per cent. of the cases; Fütterer, in 78 per cent.; Winton, in 81 per cent.; Zenker, in 91 per cent.; Courvoisier, in 91 per cent.; Siegert, in 95 per cent. and Janowski, in 100 per cent. In the senior author's series, gall-stones were present in 87 per cent. The theory of irritation as a causative factor in producing carcinoma of the gall-bladder is supported by the fact that Beadles (1896), in a study of twenty-eight personal cases of secondary carcinoma of the liver and gall-bladder, did not find gall-stones in a single instance; while in a total of sixty-three cases of secondary carcinoma gall-stones were present in only two instances.

Pathology.— Primary carcinoma of the gall-bladder is most frequently found in the fundus, the secondary site of preference being near the neck of the bladder or the beginning of the cystic duct. In other cases the entire organ may be involved. In his analysis of forty-five cases, Fütterer found the growth in the fundus in seventeen, near the opening of the cystic duct in thirteen, on the posterior wall in eight, and on the anterior wall in seven.

Most cases are of the columnar-cell type; but Konjetzni (1911) collected twenty-three instances of squamous-cell carcinoma of the gall-bladder.

Gall-bladder Removed for Cholelithiasis, Hardened Area at Neck Found on Microscopic Examination to be Carcinomatous. Female, 70 Years, Recovery. End Result not Known. Path. No. 9743. *Lankenau Hospital.*

Face p. 584

There are two types of new growth recognized, although in any case they may coexist. In one there is a cauliflower-like growth which projects into the cavity of the gall-bladder; in the second form there is a general infiltration of the walls of the organ. Although the growths usually are circumscribed in the beginning, infiltration becomes marked and nodular enlargement of the gall-bladder results. If the cystic duct is obstructed, the gall-bladder usually becomes distended; the contents usually are bloody. Perforation into the general peritoneal cavity rarely ensues, although adhesions to the surrounding viscera are common; in this way stenosis of the duodenum, pylorus, or colon, may occur. In some cases ulceration into the colon, stomach or duodenum with the formation of an internal biliary fistula may result. An external biliary fistula is very rare, except as the result of operation. Extension to the liver is found in about 50 per cent. of the cases, the processes in the liver being the result of direct extension along the biliary ducts or by the lymphatics. As noted at page 580, Beadles inclines to the view that most if not all cases of primary carcinoma of the liver arise at or near the cystic duct or neck of the gall-bladder.

Carcinoma of Gall-bladder; Cholecystectomy; Recurrence

M. S., 53 years old, married 27 years, was admitted to the German Hospital December 16, 1906.

Chief Complaint.—Feeling of distress in gall-bladder region.

Family History.—Father, mother, three sisters and one brother living and well. One sister died of pulmonary tuberculosis. Several brothers and sisters died in infancy. No malignancy.

Social History.—Two children living and well. Youngest is sixteen years old. Had one miscarriage between two living children. Forceps delivery for both children. One child born dead. One died of diphtheria eighteen years ago.

Previous Medical History.—Measles in childhood; otherwise negative. Appetite good and bowels regular before present illness.

Present Illness.—Has slowly developed a feeling of distress in the gall-bladder region with loss of appetite, weight and ambition. Gradually lost about fifteen pounds. No acute attacks of pain. Never vomited blood. Jaundice of gradual onset.

Operation.—December 18, 1906, by Dr. Deaver. Right rectus incision. Liver raised, and gall-bladder exposed. Field of operation walled off with gauze. The gall-bladder was surrounded almost completely by omental adhesions, which could be stripped off easily with gauze. The gall-bladder was enlarged, distended and full of stones of various sizes. The neck of the gall-bladder just above the cystic duct was folded so as to form a small sac pressing upon the duct. Cholecystectomy decided upon: gastro-hepatic omentum grasped and opened; cystic duct clamped and cut, and cystic vessels clamped and cut. Gall-bladder pulled free from liver

and removed. Cystic duct tied with chromic catgut, and cystic vessels with linen thread. One cigarette drain placed about cystic duct, and sewed fast with chromic gut. Several bleeding points in bed of gall-bladder clamped and tied with catgut. A piece of plain gauze rolled and placed in bed of gall-bladder to control hemorrhage, and held in place by catgut stitch. Pads and sponges removed. Viscera replaced. Wound closed in layers. (Fig. 155.

Progress.—Persistent jaundice after operation, relieved greatly after removal of second piece of gauze (Dec. 31, 1906), which had to be pulled very hard.

Discharged from the hospital, January 26, 1907.

Re-admitted to the German Hospital, April 1, 1907, less than four months after operation. Has continued to complain of weakness since leaving the hospital. Went home in a carriage and has been in bed ever since. Has had pain in the right side, with nausea and vomiting. A few weeks ago she could not keep anything on her stomach. Has had sweats with remittent and intermittent temperature. Has lost weight, but does not know how much. Has lived on liquid diet ever since leaving the hospital.

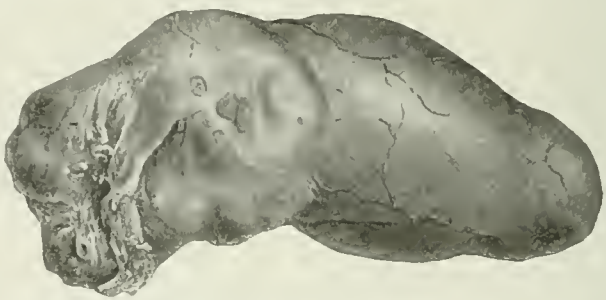


FIG. 155.—Gall-bladder Removed by Cholecystectomy. Found on Microscopical Examination to be Carcinomatous. (*Case of M. S., page* 585.)

Physical Examination.—"Lemon colored" complexion. No distinct jaundice. Heart and lungs negative.

Abdomen: Old scar and stab-wound from gall-bladder operation. Line of induration readily felt along line of drainage. Lower border of liver palpable down almost to the umbilicus. Upper border of absolute dullness about fifth interspace. Spleen not palpable or demonstrably enlarged.

Progress.—Gradual loss of weight, health and strength. At time of discharge from hospital May 14, 1907, the drainage tract had increased greatly in size and was very hard, adherent to skin.

Urine.—Shows albumin.

Stool.—Free fat, bile and occult blood negative.

Blood.—Hb., 54 per cent., W. B. C., 10,7000.

Differential Count.—Polys., 82.5 per cent.; lymph., 13; Trans., 4; Mon., 0; eosin., 0; mast., 0.5.

April 25, 1907: Hb., 45 per cent., W. B. C., 10,900.

The following case history shows the possibility of inflammation and irritation other than that caused by calculi acting as an etiological factor in gall-bladder carcinoma.

A. F., female, forty-nine years of age was admitted to the German Hospital in January, 1907.

Family history, negative. No tuberculosis or malignancy.

Previous History.—Has had seven children. Thirteen years ago had an attack of acute nephritis; four years ago an attack of pneumonia; one year ago an attack of acute appendicitis at which time there was marked pain in the upper right quadrant of the abdomen, and patient was jaundiced. *Chief complaint on admission* to hospital was profuse, foul-smelling, at times bloody, vaginal discharge.

Physical examination: large, stout, well-nourished woman, with flabby, pendulous abdomen. No pain or tenderness over entire abdomen. Vaginal examination revealed presence of a small, freely movable uterus with normal appendages. A small polyp was protruding from cervix. Two days after admission patient was suddenly seized with pain over gall-bladder region followed by rise of temperature, marked tenderness and rigidity over the gall-bladder and slight jaundice. Patient rapidly recovered from the gall-bladder attack.

Operation: March 30, 1907.—The uterus was curetted. Pathological examination showed absence of any malignance. Upper right rectus incision made. Adhesions of some standing between the gall-bladder and stomach and duodenum; new adhesions between gall-bladder and liver. Adhesions ligated and cut and gall-bladder freed. Gall-bladder aspirated and reddish-brown bile removed. Gall-bladder opened. No gall-stones found. Cystic and common ducts patulous. Mucous membrane swollen and congested. Cholecystostomy with protective gauze and rubber-dam drainage.

Subsequent Course.—Patient made a good recovery. Was relieved of all trouble until the summer of 1908, when she complained of a dull, aching pain about the incision. Pain was more or less constant, with sharp exacerbations. Has been nauseated with vomiting. During and after very sharp pains would become jaundiced. Readmitted to the German Hospital, July 17, 1909.

No jaundice. Great rolls of fat on pendulous abdomen. Sense of a mass near old scar. Tenderness on palpation.

Second Operation, July, 1909.—Upper right rectus incision. Gall-bladder region found to be a dense mass of tissue, evidently carcinoma. Numerous carcinomatous nodules on lesser omentum and about gall-bladder site. Nodule removed. Wound drained and closed to drainage. Pathological report of nodule proved it to be carcinoma.

Patient made an operative recovery and was discharged from the Hospital August 6, 1909. Subsequent history not known.

Symptoms.—These are much the same as those of primary carcinoma of the liver, especially when the tumor arises in the mucous membrane of the gall-bladder; the carcinoma that arises from the glands of the gall-bladder's mucous membrane is more apt to produce symptoms referable to the gall-bladder itself or its ducts. In the vast majority of cases the patient who presents a hard nodular tumor of the gall-bladder or liver which may be diagnosticated certainly as carcinoma, without an exploratory operation, is already beyond the help of surgery.

Treatment.—The most favorable cases are those where a thick-walled gall-bladder removed at operation is recognizably affected by carcinoma only after microscopical study. Most cases recognized as malignant during the operation prove to be too far advanced for excision to be justifiable; or if excision is done, the patients die of recurrence within a year. On the other hand, among seven cases recognized as carcinomatous only after microscopical study, Mayo had three patients who were well more than two years after operation. But whenever possible, even in cases recognized as malignant, excision of the gall-bladder, cystic duct and of the adherent surface of the liver, should be done. Palliative operation is of little use in relieving the most distressing symptoms, and in most cases merely entails upon the patient the additional discomfort of a biliary or mucous fistula. If a radical operation cannot be done, the abdomen should be closed without doing anything else. Terrier and Auvray (1901) collected sixteen cholecystectomies for carcinoma of the gall-bladder, recognized as such at operation; there were five deaths (an immediate mortality of 31 per cent.) and eleven rapid recurrences. Among eighteen operations where a portion of the liver was removed along with the gall-bladder, there were three immediate deaths (16.6 per cent.) and fourteen deaths from recurrence in from six to eight months. One patient (that of Hochenegg, already referred to at page 583) survived for three years, during two years of which time he enjoyed good health. These more radical operations had all been done since 1890. Terrier and Auvray came to the pessimistic conclusion that while the end results of radical operation for this condition were detestable, yet those of palliative operation were still more detestable. They found among fifteen cholecystomies in cases of malignant disease, that four patients died from operation, and only one survived as long as a year. Palliative operation should be done only for signs of grave cholecystitis or cholangeitis.

Primary Carcinoma of the Bile-ducts.—This occurs in men, in about 61 per cent. of cases; thus showing a marked contrast to carcinoma of the gall-bladder, which occurs in men in only about 20 or 15 per cent. of cases. In an analysis of sixty cases of carcinoma of the biliary ducts, Schüller (1901) found forty-one at the papilla of Vater, and nineteen in the common duct or the hepaticus. Rolleston found the growth situated as follows in eighty cases: In the common bile-duct, thirty-three (lower end twenty-one, mid-part, eleven); at the junction of the common bile-duct, cystic duct and common hepatic duct, twenty-five; in the common hepatic duct, eighteen; in the right

or left hepatic ducts, three; in the cystic duct, one; and in the cystic duct and lower end of the bile duct, one (Kelly, 1908). In 102 cases Donati (1909) found twenty-nine of the choledochus, thirty-four at the hepatico-cystic juncture, twenty-eight of the hepaticus, one of the cysticus, and ten not located. According to Lincini (1911) his case of carcinoma of the cystic duct was the third on record. The result of the growth of carcinoma of the ducts usually is a constriction, with dilatation of the ducts above the growth. The obstructing tumor occurs either as an annular growth which concentrically constricts the duct, or as a papillary outgrowth into the duct lumen. Wide infiltration of the ducts is rare. Carcinoma at the papilla of Vater may be of *intestinal*, *pancreatic*, or *biliary* origin. Clinically it resembles cylindrical-celled carcinoma of other portions of the intestinal tract in its slow and superficial growth, and its slight tendency to metastasis (Terrier and Auvray). Obstruction in the common duct results in distention of the gall-bladder unless this has been previously diseased and contracted. When the growth is in the hepatic duct, the gall-bladder usually is small. Enlargement of the liver occurs in almost all cases, and there may also be associated obstruction of the portal circulation and the development of ascites.

Symptoms.—In carcinoma of the extra-hepatic biliary ducts the symptoms are not marked, as a rule, until partial obstruction of the duct occurs. As in all conditions affecting the biliary tract, symptoms of dyspepsia may be noted, but usually the first symptom that calls attention to the biliary apparatus is the onset of jaundice. In most cases the jaundice develops gradually, although rare cases may show sudden icterus similar to calculus obstruction. The jaundice of carcinoma of the duct is permanent and never intermittent.

Pain, of a dull aching character, may be noted in the gall-bladder region or in the epigastrium. When a distended gall-bladder attempts to empty itself, there may be colicky pains simulating gall-stone colic.

The gall-bladder usually is enlarged on account of the obstruction, below, and is palpable in more than 50 per cent. of the cases. The liver is slightly enlarged.

The *diagnosis* must be made from calculous obstruction of the common duct. In the latter cases, the *jaundice* develops suddenly, is intense, but intermittent, the stools are not constantly acholic; in carcinoma it develops slowly, steadily increases and never becomes intermittent, no bile being found in the feces at any time after complete obstruction has once developed. In calculous obstruction, the *pain*

is sudden, severe, radiates to the right shoulder, and is accompanied by marked tenderness and rigidity in the region of the gall-bladder; in carcinoma there is only a dragging sensation, often attributed to gastric disorder. In calculous obstruction the *temperature* usually is elevated and the attack may be ushered in by a chill; in carcinoma there rarely is fever. In the former case the patient's *general nutrition* may be preserved for a long time, and the patient's appetite and digestion depend on the intensity of the colic and jaundice; in carcinoma, on the other hand, strength is quickly lost, signs of duodenal obstruction supervene, and emaciation is rapid.

An enlarged gall-bladder, in the pressure of increasing and non-remitting jaundice, is due to carcinoma in a large majority of cases; the jaundice due to common duct obstruction is accompanied by a contracted gall-bladder in 84 per cent. of the cases, according to Courvoisier (1890). Before enlargement of the gall-bladder occurs diagnosis is very difficult; but this enlargement develops so constantly as soon as obstruction is complete, that there is little excuse for failure to recognize the true lesion after the development of jaundice. Disturbance of the pancreatic functions indicated a growth at the papilla of Vater or a carcinoma of the pancreas (page 691); distinction is difficult.

Prognosis.—The disease is fatal unless all of the diseased structures can be removed by operative means. The ultimate outcome of the condition is greatly modified by the form of treatment and by the stage of the growth when operation is performed. If the diseased structures are removed early, the prognosis is fairly favorable; if the growth is not entirely removed, or if the disease is so far advanced as to be inoperable, death usually results in from six to eight months.

Treatment.—Prophylactic treatment should be instituted in all instances by removal of all gall-stones that may be in the gall-bladder and ducts as soon as their presence has been determined. The treatment of carcinoma of the *common duct* is either palliative or radical, consisting in the formation of a fistula between the gall-bladder and the gastro-intestinal tract or in a resection of the duct with removal of the growth. Resection of the duct and the various steps necessary to restore a channel for the discharge of bile into the intestinal tract have been discussed at page 508.

Removal of a growth from the **papilla of Vater** or from the **duodenal end of the common duct** may be accomplished through a transduodenal incision, as first employed by Czerny in 1901 (Schüller).

Oppenheimer (1912) collected eighteen operations of this kind; fifteen of the operations were for carcinoma with six deaths; one for benign tumor, and two for cicatricial stricture, all three successful.

Upcott also recorded (1912) a successful transduodenal excision of a carcinoma of the papilla Vater; the patient was doing well one month after operation. Outerbridge (1913), in his careful study of this subject, notes also an operation (unsuccessful) recorded by Slajner.

In cases where the growth was too extensive for transduodenal excision, a few attempts at more radical excision have been made (Halsted, Körte, Kausch). The operation in these cases was similar to the cephalic pancreatectomy of Sauvé (page 795). Halsted's patient survived for six months, but Körte's patient died in three days. The method of Cotte and Maurizot is described at p. 367. Kausch (1911) advises the following technique, which he has employed successfully, though his patient died nine months later of cholangeitis:

I. Primary operation. Cholecystenterostomy, as a temporary means to restore the patient's health. It is worth noting, however, that according to Quénu's figures (quoted in our first edition) seventeen out of twenty-one patients treated by this operation did not survive.

II. Extirpation of the growth.
1. Mobilization of the duodenum.
2. Posterior gastro-jejunostomy.
3. Section and closure of pylorus.
4. Enucleation of duodenum and adjacent pancreas.
5. Section of choledochus and pancreatic duct.
6. Duodenum is sectioned below the growth, and its distal segment is sutured over remains of pancreas, the intervening segment with the stump of the common duct and the pancreas being removed.
7. Choledocho-enterostomy.

The second stage of Kausch's operation, as described above, required four hours for its completion. In cases where the carcinoma is in the **retroduodenal portion of the choledochus,** excision of the affected portion of the duct may be attempted after mobilization of the duodenum. Oppenheimer collected eighteen such operations, ten of which were for carcinoma, with six deaths; three for calculous obstruction, with no deaths; four for benign cicatricial stricture, with two deaths; and one fatal operation for benign tumor.

After excision of the growth, the continuity of the bile-tract must be restored as already described (page 508).

Operations in the presence of obstructive jaundice from carcinoma may prove very serious undertakings on account of the great danger from

hemorrhage.[1] But in spite of the lugubrious statistics published in the first edition of this work, further experience has convinced us that operation is justifiable in any patient in whom it seems possible to afford relief, even if the operation is only a palliative one. We have had a number of patients whose last days were rendered tolerable by relief of jaundice and of much pain, as the result of cholecystenterostomy (p. 505).

[1] Quénu recommends, and has employed since 1907, the prophylactic use of antidiphtheritic serum, which is the most easily procured alien serum. He injects 20 cc. of this on the day before the operation, and since using this precaution has had only one case of postoperative hemorrhage in these icteric patients. Other measures employed as prophylaxis against bleeding have been discussed at page 436.

CHAPTER XX

INJURIES OF THE LIVER AND BILIARY PASSAGES

Injuries of the Liver

The liver is more often injured than any of the solid abdominal viscera, though scarcely so often as the intestinal tract. It is predisposed to injury (1) by its *size*, especially if this is increased by disease; (2) by its *position* in contact with the anterior abdominal wall and ribs in front, with the vertebral column behind, and with the diaphragm above; (3) by its *consistency*, being naturally inelastic, and having its friability increased by disease; and (4) by its relative *immobility*. Notable is the fact that 83 per cent. of sixty-five patients with injury of the liver, reported by Boljarski (1911), had been drinking or were actually intoxicated when they received their injuries.

In a series of 365 cases of subcutaneous injury of the solid abdominal viscera, studied by Edler (1887), the liver was injured in 189 cases, while the pancreas, spleen and kidneys, all combined, were injured only in 176 instances. Of 116 penetrating wounds, the liver was involved in sixty-five, while the pancreas, spleen and kidneys combined were involved in fifty-one cases.

Injuries of the liver may be classed as *subcutaneous* (ruptures), and *percutaneous* (stab and gunshot wounds). The relative frequency of ruptures and of stab and gunshot wounds varies considerably with the geographical location of the patients. In this country rupture is the most frequent form of injury, and this appears also to be the case in Germany, as well probably as in Great Britain and France. But in St. Petersburg Boljarski found ruptures comparatively rare.

Relative Frequency of Liver Injuries

Author	Rupture	Gunshot wounds	Stab wounds	Total
Boljarski (Russia) (1911)	8	2	55	65
Finsterer (Germany) (1912)	25	11	4	40
Tilton (New York City) (1905)	12	9	4	25

Uncomplicated injuries of the liver are much less rare than are uncomplicated injuries of any other viscus in the upper abdomen. Complicating injuries oftenest involve the pleura, diaphragm or lung; less often the kidneys, stomach or pancreas. Ruptures are much more often uncomplicated than are gunshot or stab wounds. In Boljarski's series of sixty-five cases there were the following complicating injuries:

Abdominal wall		57 cases.
Diaphragm		18 cases.
Pleura		18 cases.
Ribs		12 cases.
Stomach		6 cases.
Bowels, Mesentery, Pancreas, Spleen	each	1 case.

Prognosis.—The mortality recorded in most statistics is too low, since it is deducted from collected cases, and not from a large and consecutive series. Among isolated case reports favorable results always unduly predominate. The following classical statistics may be perused in this connection:

Cases of Injury of the Liver (No Operation)

Mayer (1872)	276 cases	59 per cent. mortality.
Otis (1876)	181 cases	62 per cent. mortality.
Edler (1887)	104 cases	57 per cent. mortality.

Edler collected in all 543 cases of liver injury, treated without operation: 189 ruptures, with 162 deaths; 289 gun-shot injuries, with 159 deaths; and 65 stab wounds, with 42 deaths; a total mortality of 66.8 per cent.

Operations for Injury of the Liver

Terrier and Auvray (1896)	56 operations	32.0 per cent. mortality.
Terrier and Auvray (1901)	42 operations	23.6 per cent. mortality.
Giordano (1902)	257 operations	33 5 per cent. mortality.

The first operation for hemorrhage from the liver was done in 1887 by Burckhardt, in a case of stab wound; the wound was packed and the patient recovered.

The prognosis of the three main varieties of liver injury is indicated by the following statistics:

STATISTICS OF OPERATIVE TREATMENT OF INJURIES OF THE LIVER

Author	Ruptures				Gunshot wounds				Stab wounds			
	Total	Rec.	Died	Mort. per cent.	Total	Rec.	Died	Mort. per cent.	Total	Rec.	Died	Mort. per cent.
Boljarski	8	0	8	100.0	2	1	1	50	55	38	17	40.0
Hagen	12	3	9	75.0					7			
Wilms	15[1]	3	12	80.0								
Finsterer	6[2]	4	2	33.0	1	1	0	0	2	2	0	0.0
Tilton				62.5				33				28.5
Thöle (all collected cases to 1909).	260	100	160	61.5	200	102	98	49	292	220	72	24.6

The great importance of *early operation* is indicated by the following statistics of Thöle:

MORTALITY AFTER OPERATIONS FOR INJURIES OF THE LIVER

	Rupture, mortality per cent.	Stab wound, mortality per cent.	Gunshot wound, mortality per cent.
1. Operation within six hours	39.5	14.5	32.2
2. Operation from seven to twelve hours	50.4	20.7	45.5
3. Operation from thirteen to twenty-four hours	66.6	33.3	50.3
4. Operation after twenty-four hours	86.3	50.0	75.4

Rupture of the Liver.—The causes are *direct* and *indirect* violence Blows, falls and crushes are the usual forms of direct injury; usually they produce stellate tears. Indirect injuries usually are effective only when the liver already is the seat of disease, though id falls upon the feet or buttocks the liver may be ruptured by "counter-stroke," or it may burst in the sagittal plane from being bent upon itself. Hubbard (1906) quotes Henzelman's statistics (1886) of 151 subcutaneous injuries of the liver, one-third of which were caused by indirect violence. Though direct violence usually acts from the front, injury may be due

[1] Four other patients, not included in the above, died of hemorrhage before operation could be done.

[2] Two other patients, not included in the above, recovered without operation. Presumably they had a subcapsular or central rupture.

to falls on the back, as in Houghton's patient (1907); to lateral pressure (Herzog, 1907), or even to muscular action (Waring). In Herzog's patient the accident occurred as the result of an obstetrical practice said to be common in the Philippine Islands: the natives place a folded cloth around the loins of the woman in labor, and one or two persons make traction upon it. This patient died, and at autopsy a rupture of the liver was found, due to the eleventh rib being forced into the liver substance.

Ruptures are much more frequent in the right than the left lobe of the liver, and upon its upper surface than upon its lower. In 182 cases analyzed by Thöle the right lobe was affected six times as often as the left.

Ruptures of the liver are divided by systematic writers into three classes:

1. Rupture of the hepatic tissue involving the capsule.

2. Separation of the unruptured capsule from the liver substance, with subcapsular hematoma.

3. Central ruptures, giving rise to separate or confluent hematomata, which may develop into cysts or abscesses.

The first class is the most important, owing to the necessary occurrence of intraperitoneal hemorrhage, with its attendant dangers. The second and third classes may prove little more serious than a contusion, unless the capsule of Glisson eventually ruptures, or unless secondary infection of the hematoma occurs.

Ruptures involving the capsule of Glisson may be single or multiple; superficial or deep; linear, stellate, or gaping; the liver substance may be pulpefied; portions of liver may be detached or the entire organ may be torn in half (Fig. 156).

Symptoms.—Immediately after the injury there usually is shock, with nausea and vomiting, followed in most cases by symptoms of internal hemorrhage. The pulse may vary considerably, being very slow very soon after the injury but soon increasing in frequency. Finsterer (1912) observed marked bradycardia in three out of eight cases of injury of the liver by contusion, and he made experimental studies to prove the value of this symptom in diagnosing rupture of the liver. Bradycardia occurs in some cases of cholemia, and Finsterer claims it is the result of the action of bile-acids, and states that even if on account of excessive hemorrhage the pulse does not become slower yet the absorption and elimination of bile-salts may be detected by appropriate tests of the urine. He claims that the pulse is the most reliable guide in those cases where internal hemorrhage is not severe,

and where rigidity of the abdominal muscles possibly may be thought to be due to a parietal injury without visceral complication. Particularly if the bradycardia develops while the patient is under observation, or if it is present at first, only to vanish as hemorrhage progresses, should injury to the liver be suspected. In one of Finsterer's patients the pulse was forty-eight, and in another fifty-two at the time of the first examination. Thus a slow pulse is of considerable diagnostic importance, though a rapid pulse does not by any means exclude visceral injury. He has collected, in all, thirteen cases of

FIG. 156.—Rupture of the Liver; from a Patient in the Episcopal Hospital. (*See Case History, page* 599.)

marked bradycardia due to this cause. Thöle found bradycardia recorded in exceedingly few of his collected cases, and thinks no significance should be attached to it.

The abdomen at first is soft and flat, and in some cases may remain so for hours; usually, however, it very soon becomes rigid and board-like, and later distended. Tenderness is more or less general soon after the injury but gradually becomes localized over the hepatic region. The respirations become shallow and thoracic in an endeavor to keep the liver at rest. As hemorrhage increases they become sighing in character. Generally there is abdominal pain, and very often pain in the back. The liver dullness may be greatly increased, and in some cases of profuse bleeding shifting dullness in the flanks

may be detected. Jaundice may occur after a day or two, especially if operative treatment has not been undertaken promptly; but as a rule it is of no diagnostic importance.

The *diagnosis* of rupture of the liver often is difficult, especially soon after the injury has been received. A history of injury in the hepatic region always should make one suspect rupture of the liver. Hubbard lays stress on the effect of heat on the abdominal muscles as a differential diagnostic sign. In simple contusions of the abdominal muscles these relax after the application of heat, while they remain rigid and the degree and extent of the rigidity increases when there is an intra-abdominal traumatism. Localization of tenderness and of spasm of the abdominal muscles often points to the organ involved. Jaundice coming on two to four days after a suspected injury of the liver, the result of absorption of bile through the peritoneum, is seen in about 20 per cent. of the cases. Jaundice appearing much later generally is due to abscess formation in the liver.

Prognosis.—The prognosis in rupture of the liver is greatly modified by the treatment instituted and the length of time elapsing between the injury and operation. Edler found that more than half of 543 collected cases of rupture of the liver died from hemorrhage within twenty-four hours following the accident. The total mortality in this series of collected cases, none of them treated by operation, was 66.8 per cent. The diagnosis, in the case of patients who recover without operation, usually is rather uncertain, and Mercadé (1902) probably was within the truth when he claimed a death rate of 80 per cent. without operation. It cannot be denied that recovery may occur without operation, especially in the cases of subcapsular or central rupture; but even in these cases a deferred operation sometimes has had to be undertaken for hepatic or subphrenic abscess or some other sequel of the original injury. According to Finsterer such cases have been recorded by Graser, Fertig and Chiari.

Much more reliable information is derived from statistics which comprise series of *consecutive cases* from one or several hospitals. Such series are presented in the following table:

Series of Consecutive Operations for Rupture of the Liver

Bartels (1904)	6 operations	83 per cent. mortality.
Boljarski (1911)	8 operations	100 per cent. mortality.
Finsterer (1912)	42 operations	69 per cent. mortality.
Hagen (1906)	12 operations	75 per cent. mortality.
Wilms (1905)	15[1] operations	80 per cent. mortality.

[1] Four other patients died before operation could be performed.

In those cases not subjected to immediate operation the prognosis is modified, naturally, by the extent of the injury, the amount of hemorrhage, and the presence or absence of infection. Usually there is extravasation of bile into the peritoneal cavity. This causes little damage if it is sterile, but when it contains bacteria, or when it is allowed to remain indefinitely in the peritoneal cavity and becomes infected by migration of bacteria from the intestines, spreading peritonitis follows. The immediate danger following the injury is from hemorrhage; the remote danger is from sepsis.

A few well-authenticated cases of *embolism* by pieces of liver tissue have been recorded. Finsterer mentions the fatal cases reported by Marshall, Hess, Schmorl, Zenker, and Williams. Schnitzler thought the pulmonary symptoms in his patient, who recovered, were due to an embolus of hepatic tissue.

The *treatment* of diagnosticated or strongly suspected rupture of the liver consists in immediate laparotomy with repair of the lesion in the liver. We believe with Nötzel (1906, 1909) that shock is not a contraindication to immediate operation. Hubbard believes that it is better to treat the shock by means of heat, morphin, strychnin, and saline solution, before opening the abdomen. As the greatest danger comes from the hemorrhage, and as the liver tissue tends to prevent collapse of the bleeding vessels, we feel quite certain it is safer to subject the patient to the added burden of an operation than to the added burden of continuing hemorrhage.

In some cases, however, death occurs so rapidly that there is no opportunity for operation. This was true in the patient whose liver is represented in Fig. 156.

Rupture of the Liver; Death

A man aged thirty-two years was admitted to the Episcopal Hospital, service of Dr. R. H. Harte, Nov. 13, 1899. He had been caught between the back of a wagon and the pole of another wagon, the pole striking the patient in the upper abdomen. There was slight discoloration of the skin posteriorly at the level of the eighth thoracic vertebra. The patient was in profound shock when admitted, unconscious, and with symptoms of internal hemorrhage. Death occurred ten minutes after admission.

Autopsy.—Peritoneal cavity full of fluid blood; left lobe of liver nearly detached from right by rupture running parallel to the great transverse fissure and about 2 cm. to its left. Only a narrow isthmus of liver tissue holds the left lobe to the right, this isthmus being at the posterior margin (Fig. 156).

The abdominal incision should be made near the median line in the epigastric region, through the right rectus muscle. The convex surface

of the liver may be reached by section of the suspensory ligament, or by resecting, the eighth, ninth, and tenth costal cartilages and drawing the ribs outward, as described at page 295. A large incision may be required, and if one from the ensiform to the umbilicus near the median line does not suffice, the best exposure will be secured by transverse division of the rectus at the level of the umbilicus, as in Czerny's gall-bladder incision (page 763).

Hemorrhage may be controlled by suture or by packing the wound in the liver with gauze. Temporary clamping of the pedicle of the liver to secure hemostasis was proposed by Pringle, according to Finsterer; and Baron (1910) devised a special clamp for the purpose.[1] One blade of the forceps is passed through the foramen of Winslow and the other in front of the gastro-hepatic omentum. Such a measure is seductive in theory, but we should be fearful of injury to the portal vein. The blood-pressure in the liver is very low, and if the surgeon does not lose his head on encountering such profuse hemorrhage, very moderate pressure with gauze on the bleeding surface is sufficient to check it.

It is perfectly feasible to suture most wounds of the liver without having bleeding along the suture tract, *provided the wounds are accessible*. In the case of lacerated wounds, where the hepatic tissue is pulpefied, it may be impossible to make sutures hold. Various methods of liver suture are described in Chapter XXIV.

Tamponade of the wound with gauze almost always is much easier than suture, but it is a method that entails a long convalescence and predisposes to infection. Boljarski found that for patients whose liver wounds had been tamponed the average stay in the hospital was sixty days. This was the case in the patient whose history is appended.

Rupture of Liver; Tamponade; Recovery

H. G., male, aged twenty-eight years, admitted to the German Hospital November 9, 1909. At 3 P.M. on the day of admission had been caught between two heavy objects crushing the lower part of the chest antero-posteriorly. *On admission*, half an hour after injury the pulse was 84, temperature 98, and respirations 28. There was very evident fracture of the tenth and eelventh ribs on the right side. Small area of dulness in both flanks. Liver dulness not obliterated. Very slight rigidity of the abdominal muscles. Peristalsis present. There was some tenderness over the hepatic region. Hemoglobin 80 per cent. One hour later there were signs of internal hemorrhage, the pulse was much more rapid, the skin and mucous membranes were becoming blanched, respirations were slightly sighing in character, and the area of dullness in the flanks was increasing. Immediate operation was advised, but was deferred by patient until 7.30 P.M., four hours and a half after the injury.

[1] McDill's experiments are referred to at page 793.

Operation by Dr. G. G. Ross, Assistant Surgeon to the Hospital. Ether anesthesia. Abdomen opened through upper right rectus. Peritoneal cavity filled with blood. Linear tear found on convex surface of right lobe of liver. The wound was 8 cm. long, very deep, and gaping. The wound in the liver was packed in layers, seven pieces of gauze being used. Blood was mopped out of peritoneal cavity, and the abdominal wound was closed, the gauze packing emerging at upper end. The patient was greatly shocked, and 800 c.c. of saline solution were given intravenously during operation; this amount was repeated three hours later.

After reacting, the patient did well. The last piece of gauze was removed on November 15, sixth day after operation. The sinus healed slowly, but was entirely closed when the patient was discharged January 21, 1910.

At the time of admission the patient's hemoglobin was 80 per cent. Two days later it was 56 per cent.

When a tampon is used it was formerly taught that it should not be removed in less than forty-eight hours, and that it should not remain in place longer than three or four days, for fear of inducing the formation of a persistent biliary fistula. But, as Boljarski (1911) pointed out, secondary hemorrhage may occur up to the sixth or seventh day, or even later; and the vast experience in wounds of the liver acquired by this writer in Zeidler's clinique at St. Petersburg should have great weight in bringing surgeons to adopt his practice. In his series of cases of liver injury the tampon was used no less than sixty times, and he made it a practice not to loosen the pack for a week, and even then to draw it out only by degrees.

The actual cautery, and the use of live steam have been recommended for the control of hemorrhage from the liver, but such measures increase the dangers of secondary hemorrhage.

Omentoplasty as an aid in controlling hemorrhage from wounds of the liver was studied experimentally by Loewy (1901), and has been used by Mauclaire in resection of the liver for tumor, and in a case of stab wound, with success. Boljarski used this method in five cases successfully: the omentum is stitched to the borders of the hepatic wound, or is stuffed into the wound, if large, and the wound margins are then sutured together over it. Boljarski used silk and ordinary intestinal needles. In three cases the abdominal wound could be closed without drainage, but in the two other patients drainage was necessary. The average stay in the hospital of the five patients was only twenty-four days, which compares very favorably with the average stay after tamponade of sixty days.

Gunshot Wounds of the Liver.—Owing to the position and size of the liver, gunshot wounds of that organ occur next in frequency to those of the intestine. In civil practice gunshot injuries of the

liver represent from 23 to 33 per cent. of all gunshot wounds involving the abdomen; but in only about 12 per cent. of such wounds is the injury of the liver uncomplicated by lesions of other viscera. But of 163 cases of gunshot wound of the liver occurring during the German War, reported by Wallace (1917), 148 (91 per cent.) were uncomplicated and only 15 (9 per cent.) were complicated by wounds of other viscera. The structures most often wounded in complicated cases are the pleura, lung, diaphragm, stomach and colon.

Gunshot Wound of Pericardium, Esophagus, Diaphragm, Liver, and Lung. Combined Operation. Death from Pneumonia.

E. F., female, aged seventeen years, was admitted to Dr. G. G. Davis's service in the Episcopal Hospital, March 6, 1909. The bullet had entered the sixth left intercostal space close to the sternum, and lodged beneath the skin of the back in the eleventh left intercostal space, about 8 cm. from the vertebral line. Shock was present, and there were symptoms of internal hemorrhage and of wound of the lung. No evidence of injury to the heart. The abdominal muscles were so rigid as to indicate, in connection with the presumed course of the bullet, that there was perforation of the diaphragm with injury of the abdominal viscera.

Operation by Dr. Ashhurst, 2.15 A.M., five hours after injury. Ether anesthesia. "Combined operation." (See p. 285.) Incision from tip of left eighth costal cartilage downward and inward to mid-line, obliquely across rectus muscle. Peritoneum opened, and a little blood found. Better exposure was secured by cutting across the eighth costal cartilage and splitting the diaphragm upward for about 7 cm. In doing this the pleura was wounded and found full of blood. The pleural opening was temporarily closed by clamp. Good exposure was now secured of the under surface of the diaphragm, the fundus and cardia of the stomach, the left lobe of the liver, and of the spleen. There was no active hemorrhage, and no wound of the stomach, colon, liver or spleen was found. The diaphragm was sutured, and the abdominal incision closed around a gauze wick. The original skin incision was then continued outward in the seventh intercostal space to the posterior axillary line, and the pleura was widely opened. There was very great dyspnea until the pleura was widely opened, whereupon the respirations became more tranquil. About 500 cc. of fluid blood were evacuated from the pleural cavity, active hemorrhage continuing. On the pleural surface of the left dome of the diaphragm was a hematoma, about 7.5 cm. long by 2.5 cm. wide, presumably a grooved wound made by the bullet. The lower and posterior margin of the lung was full of blood, but when drawn into the wound with volsellum forceps no active hemorrhage was detected, and no sutures were inserted. There was bleeding from the wound of exit in the posterior parietal pleura. This was tamponed, and the thoracic wound was closed around the gauze wick. The bullet was then removed from its subcutaneous position by another incision.

Death in fifty-three hours from double septic pneumonia.

Autopsy.—The bullet entered the sixth left intercostal space and penetrated the pericardium without wounding the heart; there was very little blood in the pericardium. On leaving the pericardium the bullet entered the diaphragm,

grooving the esophagus as the latter passed through the diaphragm. The bullet then perforated the thin margin of the left lobe of the liver, making a tunnel 2.5 cm. long. It then re-entered the diaphragm, penetrated the pleura, passed through the lower border of the lung, and left the pleura in the eleventh intercostal space. There was no blood in the peritoneal cavity and no peritonitis; no pus and little blood in the pleural cavity. Death from diffuse pneumonia involving both lungs.

The immediate effect of perforation of the liver by a bullet is hemorrhage. The extent of the hemorrhage will be modified by the size of the wound, the location and the course of the bullet, and somewhat by the period of digestion, since the hepatic blood-vessels become enormously engorged during the process of digestion. Gunshot wounds when the liver is in this condition are followed by more profuse hemorrhage than after digestion has been completed.

The *symptoms* of uncomplicated gunshot wounds of the liver are chiefly those of internal hemorrhage. Other symptoms are comparatively unimportant. Shock is not always marked. Pain may be severe or absent; it is an important symptom when referred to the right shoulder. As a rule muscular rigidity is not marked. Janndice does not occur for some days; Edler (1887) says it develops in about 20 per cent. of the cases.

The *diagnosis* depends on the position of the wound of entrance of the bullet, and the direction in which it travelled, if this is known or if it can be ascertained from the wound of exit or the site of lodgement. Usually symptoms of hemorrhage are sufficiently marked to warrant a diagnosis of wound of the liver when the course of the bullet indicates this as a probability. In most cases the complicating injuries to other viscera are of greater importance from a diagnostic and surgical standpoint than the liver injury.

Prognosis.—Uncomplicated gunshot wounds of the liver are very rare in civil life. Finsterer (1912) could find reports only of twenty-six operations in such cases, and from this series he did not exclude wounds of the pleura or slight injuries of the lung which often are negligible. Nineteen of these twenty-six patients recovered including one operated upon by Finsterer himself; and seven died, a mortality of 27 per cent. Where the liver wound is complicated by gunshot wounds of other viscera, the death rate is even higher. Among eleven consecutive cases from various German cliniques, reported by Finsterer, there were three deaths (27.2 per cent.); of Boljarski's two patients, one died (50 per cent.).

The *treatment* in all cases of gunshot wound of the liver in civil life is immediate operation. Expectant treatment might be justified in those few cases where the bullet is of small calibre, where there are

no signs or symptoms of internal hemorrhage or of complicating injuries; or where no facilities exist for operation. But in all other cases operation should be undertaken before time has elapsed for signs of peritonitis to arise. Wallace (1917) believed, from his study of war wounds (to which reference was made at p. 602), that if one could be sure the injury to the liver was uncomplicated, no operation need be done; but as this never was certain, exploration was demanded in all cases. Often the signs of internal hemorrhage are so urgent that immediate operation is required on this account. The wound of entrance should be disinfected, as advised at page 294, and the liver exposed as recommended in cases of rupture of this organ (page 599). The bleeding from the wound in the liver should be controlled by suture, packing, or omentoplasty.

Stab-wounds of the Liver.—The relative frequency of these injuries has been indicated at page 595. Among Boljarski's fifty-five cases there were thirty-two uncomplicated by injuries to other viscera.

Stab-wounds are followed by profuse hemorrhage, the bleeding being much more marked than in gunshot wounds. The *symptoms* are those of hemorrhage, possibly with symptoms of complicating injury to other viscera, as in the case of rupture and gunshot wounds.

The *diagnosis* of stab wounds of the liver is based on the history, together with the location of the external wound in the region of the liver, and on the presence of hemorrhage. It is impossible to determine positively from the symptoms alone whether or not the liver has been injured. Under ordinary circumstances the surgeon must content himself with the diagnosis of internal hemorrhage.

The *prognosis* after prompt operation is comparatively favorable. A few patients may die before opportunity for operation is afforded. Operative treatment was adopted in all of Boljarski's fifty-five patients: seventeen deaths occurred, a mortality of 40 per cent. Among the thirty-two uncomplicated cases, however, there were only four deaths (12.5 per cent.); while of the twenty-three cases complicated by other abdominal or serious thoracic injuries, no less than thirteen terminated fatally (56 per cent.).

The *treatment* consists in immediate operation. If the fact of penetration of the abdominal cavity is uncertain, the surgeon should explore the wound with the precautions advised at page 289, for the reasons there stated. In cases where the fact of penetration is assured by the prolapse of abdominal contents (a condition met with in fifteen out of Boljarski's fifty-five cases), the peritoneal cavity

may be opened at once. If the stab wound in the abdominal parietes is in a convenient location, the operative incision may be made through it. It is better when possible to operate through the stab wound than to make a new incision, although the latter of course is to be preferred if it facilitates rapid work and allows more convenient treatment of the rent in the liver. The latter should be closed by suture, packing or omentoplasty, as described at page 601.

THREE STAB WOUNDS (SUICIDAL) OF THE LIVER. SUTURE. RECOVERY.

G. S., male, Russian, aged thirty years, a tailor by occupation, attempted to kill himself at 1.30 P.M., January 5, 1912, by stabbing himself with his long tailor's shears. He was brought to the Episcopal Hospital, and admitted to Dr. Frazier's service. Examination at 5 P.M. showed that there was no shock. The pulse was 120 to 130, considerably faster than on admission, when it was about 100. He was rather pale, and quite impassive. There was no tenderness or rigidity of the abdomen. There were five stab wounds in the epigastric region. The largest, about 4 cm. long, was just to the right of the mid-line, and close to the costal margin, dividing the fibres of the right rectus muscle transversely. No prolapse of abdominal contents. No free fluid in the flanks.

Operation by Dr. Ashhurst, at 5.30 P.M., four hours after injury. Ether anesthesia, preceded by nitrous oxide. Gloved finger inserted into largest stab wound entered peritoneal cavity and recognized the liver immediately beneath the wound. Incision through right rectus muscle, close to median line, passing through the largest of the stab wounds. Free blood was present in the peritoneal cavity. The stomach and omentum were packed off with gauze, and the incision extended up beside the ensiform cartilage. On the upper convex surface of the right lobe of the liver, about 8 cm. from its anterior border, there was a stab wound, bleeding actively. By elevating the patient's lumbar spine, this wound became accessible, and was closed with one mattress suture of chromic catgut, in a curved intestinal needle. Many clots and some fluid blood sponged away from right subphrenic space. Through a stab wound in the suspensory ligament of the liver a flood of blood came from the left lobe. The suspensory ligament therefore was divided as far back as the lateral ligaments of the liver, and large quantities of fluid and clotted blood were evacuated and wiped away from above the left lobe. By depressing the liver two more stab wounds were found, both on the upper surface of the left lobe, neither penetrating to its undersurface. Only one was bleeding actively. This was closed with one mattress suture of chromic catgut (Fig. 157). The left subphrenic space was wiped dry. The stomach, gall-bladder, pylorus, omentum, and transverse colon were examined, but no further lesions were found. The suspensory ligament of the liver was then repaired by sutures, thus reattaching the liver to the diaphragm. The abdominal wound was closed without drainage. The smaller stab wounds of the abdominal wall were tamponed. The operation lasted fifty minutes, and during its performance 1000 c.c. of saline solution were administered intravenously.

Culture from the blood evacuated during operation (about 500 c.c.) remained sterile.

Recovery was slow owing to pulmonary complications. For signs of effusion in the pleural cavities (Dr. Geo. W. Norris) aspiration was done on both sides on January 22, and on the right side on January 28 and February 7. But at no time was fluid found.

The patient developed melancholia. He could not be made to eat. His

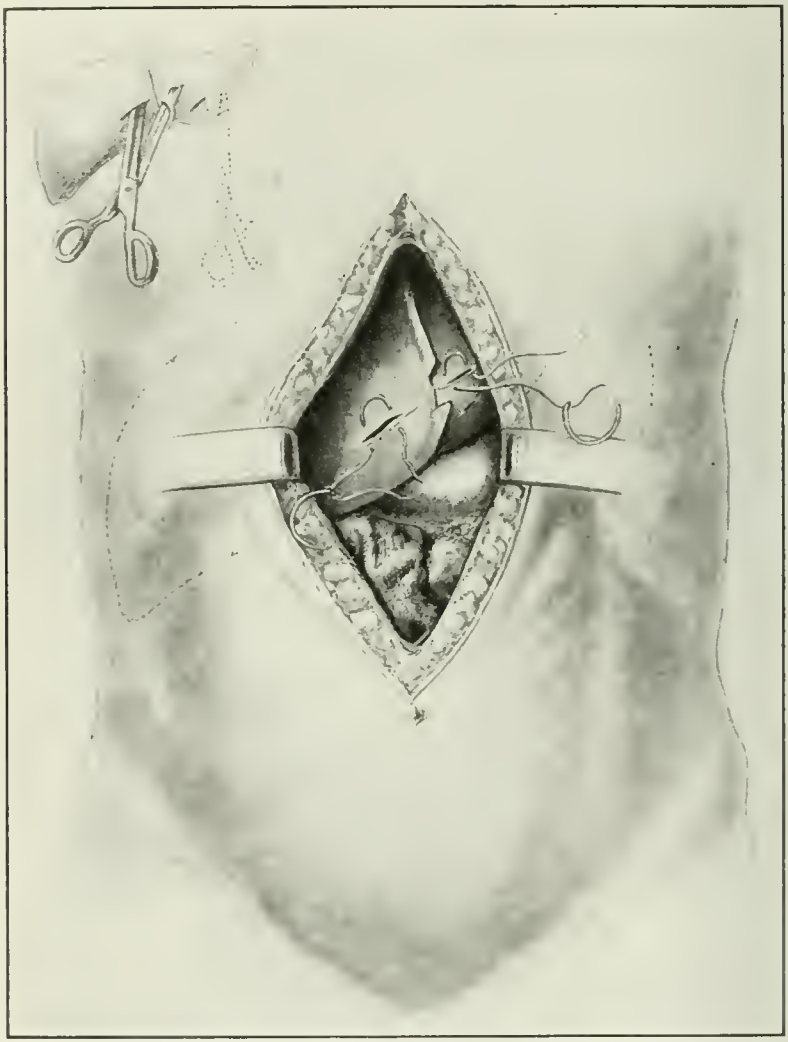


FIG. 157. — Stab-wounds of the Liver, Exposed by Division of the Suspensory Ligament. From a Patient in the Episcopal Hospital. (*See Case History, p.* 605.)

abdominal wounds were slow in healing. He was finally discharged, sound physically and mentally, on May 11, 1912, more than four months after his attempt at suicide. For the last month of his stay in the hospital he had done much work in the ward, helping the orderlies, etc.

Injuries of the Gall-bladder and Biliary Passages

Injuries to the gall-bladder and ducts occur less frequently than injuries to the liver.

Subcutaneous ruptures are the commonest. They are very seldom coincident with injuries to the liver; among the 260 cases of rupture of the liver collected by Thöle (1909), there were only two instances where the gall-bladder also was ruptured. Thöle adds thirteen cases of rupture of the gall-bladder or bile-ducts to the sixty-three cases collected in 1903 by Lewerenz. Previous disease of the biliary tract predisposes it to rupture. A distended gall-bladder such as is often seen in the presence of an obstructed cystic duct may readily be ruptured by a slight fall on the side, or by a contusion.

The fundus of the gall-bladder, which is the most exposed portion of the bile-passages, is most frequently injured. Among the sixty-three cases of rupture of the biliary passages collected by Lewerenz, the site of the lesion was

In the gall-bladder	24 cases
In the choledochus	10 cases
In intra-hepatic bile ducts	8 cases
In the hepatic duct	6 cases
In the cystic-duct	1 case
Not mentioned	14 cases

The injuring force may also cause complicating injuries of the adjacent structures, such as the liver, stomach, intestine, etc. Kilgour (1841) reported a case in which a portion of the liver with its attached gall-bladder was completely separated from the rest of the liver. Paget (1912) reported a case in which the patient was run over by a sulky. The liver had been torn from its suspensory ligament and rotated forward. The anterior edge of the liver was flattened out and adherent to the transverse colon. The gall-bladder had been torn from its bed in the liver and was suspended by a pedicle formed by the cystic duct and vessels. Bile was being discharged from a tear in the common duct. The patient recovered after two operations, death being prevented, according to Paget, by hypodermic injections of pituitary gland.

The early *symptoms* presented by rupture of the biliary passages are similar to those of other internal injuries: pain, shock, vomiting and restlessness. Signs of hemorrhage are not so marked as after rupture of the liver, as bleeding never is so profuse. Peristalsis may be present or absent. Dullness may be detected in the right flank and

later in the left flank. Vomiting in some cases has been continuous. The abdomen becomes distended and if the bile is so infectious as to cause peritonitis, the abdomen becomes tympanitic. Sterile bile, while it may give rise to very decided symptoms, does not cause fatal peritonitis. If the bile is allowed to remain in the peritoneal cavity for a considerable length of time, the intestine becomes covered with a fibrinous membrane which may be peeled off (Kehr). Kehr (1904) states that as much as twenty litres of bile have been known to collect in the abdominal cavity. As the bile is being absorbed by the peritoneum, generally after the third or fourth day, symptoms of cholemia develop.

The picture presented at a later period will be modified by the presence or absence of infection. The bile is a good culture medium and the presence of micro-organisms will give rise to a wide-spread peritonitis. Bacteria may enter the peritoneal cavity from the duodenum through the rent in the bile-passages; or if there was cholangeitis immediately prior to the injury almost invariably peritonitis will ensue.

The *diagnosis* is based on the history of injury to the abdomen or lower thorax, with a train of symptoms which points to visceral injuries. The most characteristic sign of injury to the bile-passages is the jaundice which usually appears in three or four days, due to the reabsorption of the extravasated bile. This jaundice is not noted so frequently in rupture of the liver. In a few instances the only thing suggesting injury to the biliary tract is the history of a blow or injury in the right hypochondrium; the other signs and symptoms are those following any internal injury with the exception of signs of excessive hemorrhage. The following case illustrates some of the conditions found at time of operation:

RUPTURE OF THE GALL-BLADDER. CHOLECYSTOSTOMY. RECOVERY

J. T., male, aged thirteen, admitted to the Children's Hospital of the Mary J. Drexel Home, December 29, 1906. Three days before admission he had been kicked in the upper abdomen while playing basket ball. He was unconscious for a few moments, and began vomiting one hour after receiving the injury and continued vomiting everything ingested for the next forty-eight hours. On admission patient had a temperature of 100° F., and a rapid pulse of good volume. Vomitus was greenish in color, with no odor. Urine was normal. There was no discoloration of the skin at the site of injury. Jaundice was not present. The abdomen was very much distended, the onset of this symptom having been noted thirty-six hours after the injury. There was no rigidity of the abdominal muscles. The boy complained of pain in the right hypochondrium, but there was no mass palpable and no area of tenderness could be detected. Both flanks were dull on percussion.

Operation by Dr. Deaver, December 29, 1906. On opening the peritoneum, there was a gush of a large quantity of greenish-yellow fluid, resembling bile. The entire abdomen and pelvis were filled with bile which was being discharged from a rupture in the fundus of the gall-bladder. The abdomen was flushed out with saline solution. There was no evidence of peritonitis. Cholecystostomy was performed and the pelvis was drained through a separate incision. No injury to any other viscus could be found. The patient recovered.

The *prognosis* in rupture of the gall-bladder and ducts should be favorable in all cases where early operation is performed. Of twenty-nine cases in which no operation was done, collected by Edler, twenty-two died. Septic peritonitis will follow in all cases where the bile in the peritoneal cavity becomes infected, and the prognosis under such conditions is much more grave. Even where the bile as originally effused is aseptic, it serves as such a good culture medium that its secondary infection from the intestinal tract or through the blood stream is very probable. In Courvoisier's series of thirty-four cases of subcutaneous rupture of the biliary passages, death occurred in twenty-two, in five from collapse within the first thirty hours and in seventeen from peritonitis and the toxic action of the absorbed bile. He also reported, in his fourteen cases of injury due to penetrating wounds, three deaths from collapse and six from sepsis. Uncomplicated injury of the biliary passages allows a much more favorable prognosis than the complicated cases. In the latter instances the injuries to other viscera are of far more moment than the injury to the gall-bladder or ducts.

The *treatment* consists in immediate laparotomy with repair or drainage of the injured part. If the diagnosis is uncertain we believe it is much safer to explore the abdomen than to wait until a positive diagnosis of internal injury is made from the presence of jaundice, a marked distention of the abdomen, the presence of spreading peritonitis, etc. Incision of the abdominal walls with inspection of the viscera we believe to be better surgery than removal of the fluid contents of the peritoneal cavity by means of the aspirating trocar and canula. Courvoisier reckoned that repeated aspiration might cure 33.3 per cent. of patients, while 66.6 per cent. would perish from peritonitis or cachexia. Terrier and Auvray (1896) collected seventeen cases of rupture of the biliary passages in which aspiration, at times repeated, had been performed, ten of the patients recovering. Unless the rent in the bile-passages is very small so that it may be closed by plastic exudate in a short time, aspiration will not remedy matters as more bile will be constantly poured into the abdomen and absorbed. Aspira-

tion can only remove the bile from the peritoneal cavity; it does not effect a closure of the rent in the injured structures.

When the opening in the gall-bladder is small, it may be closed and the external wound closed without drainage after evacuation of most of the fluid found in the abdomen. Flushing or irrigation of the peritoneal cavity is to be condemned. When the rupture is of considerable extent, the gall-bladder should be drained; or if the gall-bladder has been badly torn or shows the presence of marked disease, it should be removed. When the common duct is ruptured the opening, if small, may be closed; if the rent is large, drainage by means of a rubber tube should be instituted. If the entire lumen has been severed, the posterior wall of the duct may be sutured, with drainage by means of a T-shaped tube from the remaining opening in the anterior portion; or some form of anastomosis between the bile-tract and intestine may be done. In all cases, however, in which there is evidence of cholecystitis or cholangeitis, drainage should be instituted, and all attempts at radical operation postponed until the patient becomes convalescent.

Retroperitoneal Ruptures of the Bile-ducts.—This subject was referred to at p. 453 in connection with biliary peritonitis.

Penetrating wounds of the gall-bladder are quite rare. Thöle gives references to seventeen cases in which operation was done: thirteen patients recovered, and four died, a mortality of 23.5 per cent. He found no published cases of open wounds of the bile-ducts.

CHAPTER XXI

SURGERY OF THE PANCREAS

Historical.—The term pancreas, from the Greek πᾶν κρέας, meaning literally "all flesh," is found in the writings of Galen; and it was thought by Haller that there was a reference to it in the apocryphal work of Hippocrates (περι ἀδένων). According to Galen, an anatomical controversy arose over the pancreas between Herophilus and Eudemus (fourth century B. C.), the latter of whom stated that a fluid emptied out of the pancreas into the bowels and aided in digestion; yet this interesting fact was entirely forgotten under the influence of the humoral pathology of Galen (second century A. D.), who followed the teaching of Aristotle that the pancreas acted as a cushion or buffer for the stomach and the neighboring vessels. Even the early anatomists, such as Vesalius, Fallopius, and Bauhinus knew nothing of the function of the pancreas, though it is said that Alberti (1578) and Heurnius (1599) made it the subject of dissertations. Wirsung (1642) described the duct which bears his name, but his contemporary, Asellius, confused the pancreas with the retroperitoneal or mesenteric lymphatics; while some held that it acted as a filter for the secretions of the liver and the spleen.

In 1663 Regner de Graaf published a work on the nature and use of pancreatic juice; he was also the first to describe pancreatic calculi. This was long before Santorini's work, which, however, was not published until 1775, thirty-eight years after the author's death. During this period (seventeenth and eighteenth centuries) the pancreas was associated in the medical mind with such diseases as melancholy, hysteria, and epilepsy. Such ideas were soon abolished, however, by the physiological and pathological discoveries of the nineteenth century. Claude Bernard described the pancreatic ferment in 1856, and the pathological anatomists of the day, Cruveilhier, Rokitansky, Virchow, v. Recklinghausen, and later, Orth, Langerhans, and Dieckhoff, laid the foundation of our present knowledge of pancreatic diseases by studying the gland *post-mortem*.

Hemorrhage in connection with diseases of the pancreas, the so-called "pancreatic apoplexy," was first noted in 1866 by Spiess.

Fat necorsis accompanying acute pancreatitis was first described by Balser, who noticed it first at autopsy in 1879; but the publications of Fitz, beginning in 1889, were the first to make the profession realize the importance of the acute affections of the pancreas.

Though the connection between pancreatic diseases and diabetes was suggested by many writers[1] (Chopart, 1791; v. Recklinghausen, 1864; Lancereaux 1877), it was not firmly established until the publication in 1889 of v. Mering's and Minkowski's experimental work.

Gussenbauer, in 1882, was the first to operate successfully by laparotomy in a case of pancreatic cyst,[2] though Lücke (1867) and Rokitansky (1881) had previously done such operations unsucessfully, and Thiersch (1881) and Kulenkampff (1881) had opened pancreatic cysts with success, in two stages. But the true Father of Pancreatic Surgery is Nicholas Senn, who in 1886 published his experimental and clinical researches, with the object, as he modestly stated, of laying a foundation for the rational treatment by surgical means of some of the diseases of the pancreas. In 1896 Riedel first recognized chronic interstitial pancreatitis at operation; but it is chiefly to the teaching and example of Mayo Robson, from the year 1900 on, that modern pancreatic surgery owes its inspiration and guidance.

Congenital Anomalies, Displacements, etc.—The pancreas very occasionally may be entirely *absent;* three such cases, associated with other congenital anomalies incompatible with life, are mentioned by Lancereaux (1899). On the other hand, in a certain proportion of bodies there are so-called *accessory pancreases.* Letulle (1900) found this condition six times among two hundred bodies examined postmortem, while Opie (1903) found it only ten times in eighteen hundred autopsies. It is probable that systematic search might reveal one or more accessory pancreases in a fairly large proportion of autopsies. These accessory glands are usually of small size, comparable to a pea or bean, or even smaller, being very seldom so large as a nut; they are situated in the walls of the various portions of the alimentary tract, usually in the muscular coat; and they nearly always have clearly recognizable ducts. In a few instances islands of Langerhans have been present. In the cases collected by Robson and Cammidge (1907), the accessory pancreas was situated in the stomach in eleven instances, in the duodenum in six, in the jejunum in eleven (nearly all close to its

[1] Cawley (1788) is quoted (by the name Cowley) by many recent writers on the pancreas as one of the earliest to recognize this condition. Though the pancreas of his patient was full of stones, he distinctly states his belief that the seat of diabetes is in the kidneys, and the lesions of the liver and pancreas are the result, not the cause of that disease.

[2] Pancreatic cyst was first studied by Engel in 1841.

duodenal end), in the ileum in five (and always in association with an intestinal diverticulum, not Meckel's), and in a congenital umbilical fistula in one case. The accessory pancreas may occur as a single, isolated, glandular mass, or several masses may be distributed along the gastro-intestinal tract. Several cases are on record of an accessory pancreas at the duodenal opening of the duct of Wirsung; and in almost all individuals, lobules of pancreatic tissue surround the duct of Santorini as it passes through the duodenal wall (Opie). Usually all accessory glands are found at autopsy to be more or less diseased, and as the total amount of glandular tissue is very small it has been questtioned whether it can in any efficient way supplement the functions of the pancreas itself.

That portion of the head of the pancreas which lies furthest to the patient's left sometimes becomes more or less separated from the rest of the gland by the superior mesenteric vessels, which emerge just above it from between the body of the pancreas and the transverse duodenum. This partially isolated portion of the head is sometimes described as the *Pancreas Minus*, but is not truly an accessory gland. Other portions of the pancreas occasionally are more or less isolated from the remainder of the gland, this condition being described by Glinski (1901), who studied the subject carefully, as *Pancreas Divisum*. That portion of the pancreas associated with the duct of Santorini, which arises as a separate outgrowth from the posterior wall of the duodenum, is that which is most often separated from the rest of the gland, which is developed as a twin outgrowth from the duodenal wall. Normally it coalesces with the portion developing around the duct of Wirsung; and in the adult the body and tail of the pancreas are more apt to drain through the duct of Wirsung than through the duct of Santorini around which they were primarily developed.

The head of the pancreas may surround the duodenum completely, constituting the so-called *Ring-formed Pancreas* (*Pancreas Annulare*); Robson and Cammidge refer to nine such cases. In a few instances this rare condition has simulated, soon after birth, infantile stenosis of the pylorus; Vidal (1905) successfully resorted to gastro-enterostomy in such a patient three days after birth. Symptoms may not develop until adult life, when chronic pancreatitis, carcinoma, etc., affecting such an abnormally situated pancreas, may produce occlusion of the duodenum, which can be distinguished from pyloric stenosis only with the greatest difficulty. Lerat (1910) treated such a case, in a woman aged forty years, by partial pancreatectomy.

Displacement of the pancreas is very unusual, owing to its fixed retroperitoneal position. Sappey, according to Körte (1898), claimed that tight lacing might eventually lead to displacement of the pancreas, by compressing the lower thorax. The tail is the least well fixed portion, and occasionally is dragged from its moorings by a wandering spleen (Helm and Klob, 1856; Estes, 1882; Runge, 1895). Solid tumors of the pancreas may also cause its displacement; in one-fourth of the seventeen cases studied by Finney (1910), the tumor was freely movable.

Among the 276 cases of diaphragmatic hernia collected by Lacher (1880), the pancreas was among the organs displaced into the thorax in no less than twenty-seven instances; while this was the case in two out of the twenty-six cases of congenital diaphragmatic hernia collected by O. Mayor in 1891, which had been reported since the appearance of Lacher's paper. Such cases have very little surgical interest, so far as the pancreas is concerned, as any symptoms which may arise from interference with its function are sure to be overshadowed by the changes induced in the stomach and intestines.

The pancreas has been found three times in a *congenital umbilical hernia*, according to Körte (1898); who also states that E. Rose and Rahn have each found it in a similar hernia in adults. Finally, reference must be made to the remarkable case observed in 1805 by Baud in which the pancreas formed part of a complicated case of *intussusception*. Guibert in 1829 described a somewhat similar case.

Infections of the Pancreas

Pathogenesis.—*Infection of the pancreas* may occur in any one of four ways: (1) through the blood stream; (2) along the excretory ducts of the pancreas; (3) through the lymph-channels; and (4) by contiguity from neighboring structures.

1. *Infection through the blood* is considered rare. The pancreas is not situated as is the liver in relation to the portal circulation, and thus is not constantly inundated with hordes of bacteria from the intestines as we are now led to believe is the case with the liver (see page 417). The pancreas is thus almost immune from infection through its venous channels, except by *retrograde embolism in portal thrombosis;* a case, possibly of this nature, was reported in 1885 by Musser, though it is not impossible that there was here an ascending sialodochitis. The pancreas occasionally is the seat of *pyemic abscess*, as in the case reported by Roddick (1869) and as in one patient with appendicitis,

under the care of the senior author; or is diseased in other general infections; indeed the experimental and clinical facts recently adduced in support of hematogenous infection, by Abrami, Richet and Saint-Girons (1910) make it probable that this source is less rare than hitherto has been supposed. Macaigne (1894) observed a case of pneumococcic peritonitis with an abscess of the pancreas from which a pure culture of the *pneumococcus* was obtained; and though he expresses the conviction that the infection of the pancreas was received by way of its ducts, it seems quite as likely to have been hematogenic. S. Phillips (1908) observed a case of *scarlet fever* with pancreatitis and parotitis; and the occurrence of lesions in the pancreas as well as in the suprarenal glands, during the course of scarlet fever has been carefully studied by Tixier and Troisier (1912). The association of *epidemic parotitis* (mumps) with pancreatitis is now well recognized, and though Fitz (1889) wrote that "it may be safely stated that there is no reason for admitting the existence of a metastatic pancreatitis secondary to inflammation of the parotid gland," the belief is now very general, and we believe it is well established clinically, that such an affection does occur; but the pathogenesis of this metastatic pancreatitis is no more easily explained than the similar involvement of the testicles or ovaries. It was Schmackpeffer who, in 1817, first called attention to the fact that the pancreas might be involved in mumps. Gordon Sharp (1908) points out that pancreatitis may precede as well as follow the parotitis. Ordinarily the symptoms of acute pancreatitis occur on the third or fourth day after the parotid swelling has reached its height; and the parotid may undergo resolution so soon as the abdominal symptoms appear. Sharp likewise calls attention to the fact that during an epidemic one patient in a household or in an infected area may show parotitis, while another patient may have only pancreatitis; but he contends that both are due to one and the same cause. If parotitis occurs in the course of pancreatitis, it seems much more reasonable to suppose, unless there is an epidemic of the disease, that the infection reaches the parotid along its duct, from the mouth, as in typhoid fever, and as occasionally observed after etherization.

From the observations of Simonin (1903), pancreatitis appears to be a rare complication of mumps; in 652 cases of the latter disease he carefully examined the abdomen for signs of pancreatic involvement, but found it only in ten cases (1.53 per cent.) Other observers, however, have found it much more prevalent in certain epidemics, as was demonstrated in the table published in the first edition of this

work. This table, which gave all the cases we could find, up to that date, of pancreatic symptoms occurring in patients with mumps, indicated that 61 cases were on record; and that 51 of these were known to have occurred in a total of 789 cases of mumps, or an incidedce of 6.5 per cent. So far as is known, the case of Lemoine (1905) is the only one which terminated fatally. In Edgecombe's patient (1908) the urine, examined by Cammidge himself, gave a positive "pancreatic reaction."

In this connection it is interesting to note that *diabetes*, presumably of pancreatic origin, has occasionally followed the mumps (Harris, 1899).

Influenza, *syphilis*, *tuberculosis*, *malaria*, *typhoid fever*, etc., are occasionally accompanied by *chronic pancreatitis*, which is due to the local action of the infecting organism when this circulates in the blood, or, as is probably more often the case, to the toxins produced by the bacteria which are localized elsewhere in the body. Warthin (1916) found not one normal pancreas at autopsies on 150 syphilitics, and believes syphilis is the most common cause of chronic interstitial pancreatitis. Sailer and Speese (1908) succeeded in producing focal necroses in the liver and pancreas of guinea-pigs by injecting blood serum from dogs affected with experimentally induced acute pancreatitis. Injections of normal serum had no such effect. It was also found possible to produce a high degree of immunity by gradually increasing the doses of the toxic serum. Egdahl (1907) referred to cases of *acute pancreatitis* following or associated with the following affections: syphilis, typhoid fever, malaria, emboli, furunculosis, bronchitis, heart-disease, pulmonary tuberculosis, and appendicitis. Hirschfeld (1909) studied in some detail the relation of general infections to diseases of the pancreas.

In *arteriosclerosis*, which may be considered an intoxication, if not a toxemia, the pancreas is quite constantly affected by a chronic indurative inflammation (cirrhosis), and the arteries are often the seat of miliary aneurisms (Lancereaux, 1899), a fact which sometimes is held to explain the frequency of hemorrhagic lesions in this organ.

Alcoholism possibly may transmit a non-bacterial inflammation to the pancreas through the blood stream, either directly, or, as is probably much less frequently the case, by chronic passive congestion through hepatic cirrhosis. It is taught by Opie (1908) that both hepatic and pancreatic cirrhosis are due to the same cause, and that the pancreatic condition is seldom if ever caused by obstruction of the portal circulation alone. Another condition in which the pancreas

is quite constantly involved in a cirrhotic process is that described in 1899 by v. Recklinghausen as *hemachromatosis*, which in its later stages frequently is accompanied by the *diabète bronze* of the French.

2. *Infection through the Ducts.*—It is usually taught that in the pancreas infection most often occurs by continuity of structure upward from the duodenum, through the papilla of Vater and along the duct of Wirsung. But though numerous experiments, which will be mentioned presently, prove that this avenue of infection is quite possible, other experiments and certain anatomical facts tend to make us question its frequent occurrence.

Opie (1903) found that the valve-like folds within the diverticulum of Vater prevent the regurgitation of material from the duodenum into the duct of Wirsung, and that if, after death, fluid is forced under considerable pressure into the duodenum, tied above and below the pancreas, none enters the duct. Archibald (1919) reports that he tried the experiment, the solution being under a pressure of 1000 mm. of water for an hour; but none entered the ducts. Moreover, in only five out of 223 cases examined did Truhart (Böhm, 1904) find bacteria present in the normal duct of Wirsung;[1] and the singular freedom of the duodenum from bacterial life has frequently been pointed out. As has been seen already (page 417), biliary infection usually occurs primarily through the portal blood stream, being thus a descending infection from the liver; and infection of the ducts, unless these are obstructed by calculi, does not occur to any marked degree because the bile is more or less constantly passing through them, and thus clears them of infectious material. But the gall-bladder, where the bile is prone to become a stagnant pool, is frequently the site of infection, and this infection is to all intents and purposes primary there, though really having its origin in the infected bile secreted by the liver. Now in the pancreas there is no such stagnant pool as is found in the gall-bladder; the pancreatic juice is more actively bactericidal than the bile (Remedi, 1905); and blood infections of the pancreas are rare; rare also, therefore, is a descending infection of the pancreatic duct.

But there is this to be said in favor of the occurrence of an ascending infection of the pancreatic ducts—that any obstruction to the outflow of pancreatic juice will predispose it to infection, and may make quite possible a pancreatic infection arising in the mildly infectious bile passing through the ampulla of Vater. Such obstruction of the duct of Wirsung frequently occurs in the case of gall-stones impacted in the

[1] The existence of anaërobic bacteria in the normal biliary and pancreatic ducts has been emphasized as a cause of infection in cases of obstruction by Gilbert and Lippmann (1908).

lower end of the common bile-duct. Besides such factors as these, it must not be overlooked that the accessory pancreatic duct (Santorini) must sometimes be taken into consideration as a possible avenue of infection. It is claimed by Desjardins (1905) that, while the normal current of pancreatic juice in the duct of Wirsung flows toward the duodenum, the flow in the duct of Santorini is indifferently either toward the duodenum or away from it toward the intraglandular anastomosis of the two ducts; so that in this way, he asserts, micro-organisms from the duodenum easily and frequently ascend by the duct of Santorini, and meeting the outflowing current in the main duct are carried back through the head of the pancreas, thus doubly infecting the area enclosed between the two ducts. This area has been named by Desjardins the "triangle of infection." This theory takes little account of the supposed bactericidal action of the pancreatic juice, or of the relatively mild infectiousness of the duodenal contents; and fails to explain cases of pancreatic infection in which the duct of Santorini is not patent, or does not anastomose with the duct of Wirsung. Schirmer (1893) found the duodenal orifice of the duct of Santorini permeable in only a little over half of the cases (about 100 in number) examined by him. In only forty-eight out of 100 specimens dissected by Opie, was the duodenal orifice of the duct of Santorini permeable to injected fluid, while in forty-two cases its duodenal orifice was closed, the duct of Santorini appearing merely as a branch of the main duct; and in ten cases the two ducts were not in anastomosis at all.

But even if we acknowledge, as it seems we are bound to do, that ascending *infection* of the pancreatic duct is rare, there nevertheless remain experimental and clinical proofs that disease of the pancreas does undoubtedly arise from the *retrojection of bile* into the pancreas, from its chemical action, even if the bile is not actively infectious. Opie's much quoted case (1901) is an interesting example: at autopsy the cause of acute pancreatitis was found to be a small biliary calculus lodged at the duodenal orifice of the ampulla of Vater; the calulus was so small that it did not obstruct the orifice of the duct of Wirsung, but by occluding the common outlet of this duct and of the common bile-duct, allowed retrojection of bile to occur into the pancreatic duct. Similar results have been obtained experimentally by Carnot (1898), Flexner (1900), and several other investigators (accounts of whose work are given by Flexner), by injecting various substances directly into the duct of Wirsung. Flexner showed subsequently (1906) that when bile is injected into the pancreatic duct, it is the bile-salts

which are the destructive agent, and that the more colloid material the bile contains the less intense is the inflammation produced by it. Flexner therefore, suggested that as in chronic inflammation of the biliary passages (as in cholelithiasis) there is a loss of diffusible salts and an increase of colloid material, ascending infection of the pancreatic duct should in such cases lead to chronic rather than acute pancreatitis; and that this is actually the case will be pointed out in a subsequent section. Nordmann (1913) found if he closed both pancreatic openings in dogs, and injected bacterial cultures into the gall-bladder, that all the dogs died of acute pancreatitis; if only the common opening of the bile and pancreatic ducts was closed, no pancreatitis developed, as the pancreas drained itself through its accessory duct; but the dogs died of cholangeitis.

Archibald, in numerous able papers since 1910, has maintained the theory of the "retrojection of bile" as the most rational explanation of the pathogenesis of pancreatitis; and has published numerous experimental researches and clinical cases which lend support to his views. He notes, as have other surgeons, that as operative experience has increased, so *pari passu* have increased the number of cases of pancreatitis, acute or chronic, in which no gross lesion (calculi, cholecystitis, etc.) of the biliary tract could be demonstrated; indicating that neither a mere descending infection nor yet the impaction of a calculus in the ampulla of Vater was a sufficient explanation in all cases. The explanation which Archibald offers (1912, 1913, 1919) is a spastic contraction of the sphincter known by the name of Oddi (1887) surrounding the duodenal orifice of the common duct. The importance of this sphincter was scarcely recognized by surgeons until Archibald began calling attention to it, though the existence of the sphincter was well known, and it was mentioned in our first edition (1909, Vol. I, p. 39). He thinks it possible that this spasticity of the sphincter of Oddi may be aroused by hyperacidity of the duodenal contents; and he suggests that both in alcoholic individuals and in those with duodenal ulcer (in both of which classes pancreatitis is rather frequent), the duodenal contents may remain acid for a long time. Chemical researches (1920) under his direction, indicate that in *infected* bile the proportion of bile salts (the active agent in producing pancreatitis) to mucin is increased from six to twelve fold over that present in normal bile.

Experimentally chronic pancreatitis has been produced (*a*) by obstruction of the pancreatic duct by a ligature; (*b*) by injecting into the pancreatic duct attenuated cultures of various micro-organisms,

modified bile, etc.; (*c*) Carnot produced chronic pancreatitis from an ascending duodenal infection by the ingenious plan of fixing a thread in the duct of Wirsung and carrying it through the ampulla of Vater into the intestine, where it was allowed to hang free. But such experiments as these, as very justly remarked by Maugeret (1908), imply such actual traumatism to the pancreas as to make the interpretation of the results obtained very hazardous. Whether obstruction alone is an efficient factor in producing pancreatitis is a question very difficult to decide, because as soon as the pancreatic secretion becomes stagnated the virulence of the anaërobic bacteria (which, as already remarked, may be considered normal inhabitants of the ampulla of Vater and lower pancreatic ducts) is markedly increased and the subsequent changes may be attributable largely to their action. As Ebner (1907) tersely expressed it, the results of obstruction are first mechanical, then chemical, and then bacterial.

Intestinal parasites have been observed occasionally in the duct of Wirsung, according to Lieutaud and others (cited by Carnot, 1908). Muroya (1912) found ascarides encapsulated in the pancreas.

Infection through the Lymphatics.—Largely by exclusion, apparently, investigators have been led to believe that chronic infection of the pancreas in a majority of cases is due to invasion of its substance by way of the lymph-channels. This view is held by Prof. Thiroloix, and was developed and ably supported by his pupil Maugeret (1908), who urges that as the efferent lymphatics from the gall-bladder and those from the pancreas anastomose around the head of the pancreas, this part of the gland is in this manner easily invaded directly from the lymph-nodes or lymph-channels. For this pathological state we adopted (1912) the term *pancreatic lymphangeitis*, proposed by Arnsperger (1911). It is a condition which precedes true interstitial pancreatitis, and which is curable by proper surgical treatment. According to this view, which we believe to be correct for the great majority of cases previously classed together as chronic pancreatitis, it is still the infected bile and the diseased gall-bladder which must be incriminated as the "*fons et origo mali*," though we have also pointed out the possibility of pancreatic lymphangeitis occurring as the result of duodenal and even gastric lesions (page 663). It has also been suggested that though an ascending catarrhal infection along the pancreatic duct is rare, yet micro-organisms may and frequently do travel up *in the walls* of the pancreatic ducts, as they are known to do up the walls of the choledochus, even after this has been ligated. The common bile-duct is imbedded in the head of the pancreas in from 60 to 95 per

cent. of cases (Helly, 1898, Ebner, 1907, Kehr, 1909), so that any affection which injures the walls of the choledochus will be very liable to spread to the surrounding pancreatic tissue through the lymphatics. On such grounds has been explained the very frequent occurrence of chronic pancreatitis as a sequel of common duct calculus; but Maugeret contends that this frequent association of pancreatitis with common duct calculus is susceptible of another interpretation, namely, that the calculus is arrested in the common duct by the narrowing of this channel consequent upon the previous existence of a pancreatitis, which in this as in other cases is caused not by a calculus in the common duct, but by infection in the gall-bladder; and she further calls attention to the extreme rarity of such local lesions in the common duct (suppurative or ulcerative angeiocholitis) as could give rise to pancreatitis by contiguity.

The subject of *pancreatic lymphangeitis* is discussed at page 659.

4. *Infection by contiguity* is of infrequent occurrence, unless propagation of infection from the walls of the common duct, which has just been mentioned, is to be included here. The stomach is the organ which is most often at fault in these cases, ulcers perforating into, and cancers becoming densely adherent to the pancreas, and setting up a localized inflammatory reaction. Splenic abscess may involve the tail of the pancreas; pyonephrosis, on either side, but usually the left, sometimes has transmitted its infection to the adjacent portion of the pancreas; and the head of the pancreas is not seldom invaded by tuberculosis or other infection localized in the subpyloric or retropancreatic lymph-nodes.

Pancreatic Calculi.—Although concretions in the pancreas were described (de Graaf, 1663) long before cysts (Engle, 1841), pancreatic apoplexy (Spiess, 1866), or fat necrosis (Balser, 1879), they occupy, when compared with biliary calculi, a very insignificant place in the surgery of the pancreas. Opie found only two instances of pancreatic calculi among 1500 autopsies, and thinks that the figures of Giudiceandrea (two in 122 autospises) much exaggerate their frequency. Lazarus, in 1904, was able to find records of only eighty cases of pancreatic calculi. Einhorn (1916) has observed two cases, and a few additional cases are on record.

Pancreatic calculi occur nearly five times as often in men as in women, according to Lazarus, who found the sex mentioned in fifty-seven cases, forty-seven out of which were found in men. Lazarus points out that both infection and stasis of the secretion are necessary for the formation of pancreatic concretions. Guidiceandrea (1896)

found bacteria in the center of pancreatic calculi, and it is interesting to note that the same experience in regard to salivary calculi, which closely resemble those of the pancreas, has been recorded by Galippe (1893).

Pancreatic calculi usually are multiple, the largest being near the orifice of the main duct, and the others, perhaps hundreds in number, scattered along its entire length and even in the finest branches of the pancreatic ducts (Fig. 158). Occasionally star-shaped or branched calculi are found. The largest stone ever recorded, according to Lazarus, was that of Schupmann, which measured ½ inch by 2½ inches, and weighed, according to Villar (1905), 200 grams. Pancreatic calculi are composed almost entirely of calcium carbonate or calcium phosphate, are not crystalline, and, because of the narrowness of the pancreatic

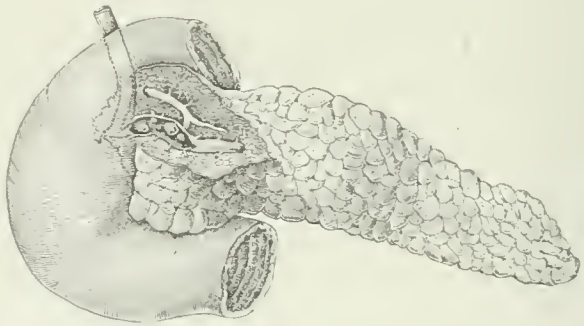


FIG. 158.—Pancreatic Calculi. (*From a Specimen in the Museum of the Lankenau Hospital.*)

ducts, they are very rarely faceted except at their ends. Usually they are white. Solitary stones, which are quite rare (four among twenty-two cases, Lazarus), are more apt to be composed of oxalates. The nucleus of pancreatic calculi probably is derived from the interaction of the desquamated epithelium lining the ducts with the secretion which has been modified as the result of infection; the result of this interaction is the precipitation of inorganic salts (which are not found in normal pancreatic juice); and new layers are formed as in other calculi (biliary, renal) by the deposit of calcium or magnesium phosphates and carbonates, or even of cholesterin.

While pancreatic calculi are in the first instance the *result* of disease of the pancreas, they become when once formed the *cause* of further structural alterations. By damming up the pancreatic secretion they lead in time to a condition of very marked sclerosis of the gland. Of the eighty cases of pancreatic calculi collected by Lazarus, thirty-six (45 per cent.) presented also diabetes, or at least glycosuria.

Carcinoma of the pancreas, however, rarely is associated with the formation of stones. Like calculi of other organs, those of the pancreas occasionally ulcerate their way into other viscera, or even into the peritoneal cavity. Galliard (1880) recorded the case of a patient who was found at autopsy to have a pancreato-gastric fistula, one pancreatic calculus lying free in the stomach, and others being still within the pancreas. In Clayton's patient (1839), who died with symptoms of internal hemorrhage, a calculus was found to have ulcerated out of the pancreas into the general peritoneal cavity, opening a blood-vessel; other calculi were found within the ducts of the pancreas.

According to Desjardins (1905), the rarity of pancreatic calculi and the frequency of gall-stones are to be explained by inherent differences between the biliary and the pancreatic systems. He holds, and Quénu and Duval (1905) agree with him, that the same infection arising in the intestine and traveling up the bile and pancreatic ducts will in the former situation induce a stone-forming catarrh, but in the pancreas will cause a chronic interstitial inflammation.

The symptoms, diagnosis and treatment of pancreatic calculi are considered at page 685.

Pancreatic Diabetes.—The connection between pancreatic disease and diabetes has been recognized since the time of Cawley (1788), though this observer distinctly stated his opinion that the numerous calculi which were found in the pancreas of his patient were the result, not the cause, of the diabetes. In 1858 Claude Bernard discovered that puncture of the floor of the fourth ventricle caused glycosuria, and for years the nervous theory of the origin of diabetes seemed to rule the medical world. It was not until 1877 that the theory of pancreatic diabetes as a clinical entity was formally propounded by Lanceraux; and the theory was first firmly established as fact by the experimental work of v. Mering and Minkowski, in 1889. Of late years the number of cases of diabetes, in which no lesions are to be found in the pancreas, appears to be steadily decreasing. This increase in the number of cases of pancreatic diabetes is due largely to more careful examination, especially to microscopical study of the pancreas post-morten. According to Cammidge (1908), some observers claim that all cases of diabetes are pancreatic in origin, but this gland was proved at fault only in about 88 per cent. of the 288 cases of diabetes mellitus collected by Opie in 1908. In 6 cases of diabetes, where the pancreas was examined at autopsy by Warthin (1913), the patients were syphilitic, and he regarded the pancreatic lesions as being syphilitic in origin.

The changes in the pancreas which lead to diabetes are believed to be confined to the islands of Langerhans. The secreting parenchyma or the interstitial tissue of the gland may be very extensively diseased, even almost entirely destroyed; yet it seems an established fact that so long as a certain proportion of the islands of Langerhans remains intact, glycosuria does not occur. It is, moreover, apparently proved that the development of glycosuria in disease of the pancreas is to be attributed to interference with the internal secretion of the gland; for it has been found (Minkowski), if a portion of the pancreas is successfully transplanted into the subcutaneous tissues, being severed from all its nervous connections, that the entire portion remaining in the abdomen may be removed and that glycosuria will not develop until the transplanted portion also is removed, or until it becomes atrophic.

It is evident, therefore, that the cause of glycosuria and of the accumulation of sugar in the blood is dependent upon some influence which the pancreas exerts by way of the blood or lymph stream. Two theories are proposed to explain the means by which these changes are brought about: (1) *The auto-intoxication theory*, by which it is assumed (Robson and Cammidge, 1908) "that the cells of the pancreas normally destroy, or modify, some toxic substance, produced in other parts of the body, which interferes with the utilization of sugar by the tissues;" (2) the theory that the pancreas contributes some *activator substance* to the circulation, which assists a glycolytic enzyme produced by other tissues of the body. The second theory is that which has received most support of late.

The relation of the *adrenals*, and of the *hypophysis cerebri* to pancreatic activity has been too little studied, and is still much too obscure a subject for any authoritative statement to be made at this time. It is perhaps sufficient to recall the existence of cases of pancreatic infantilism; to note that a diminished function of the anterior part of the hypophysis is productive of similar changes (von Eiselsberg, 1910); that certain cases of acromegaly (*hyperpituitarism*) have been associated with diabetes (Opie); and that the local action of adrenalin upon the pancreas causes temporary glycosuria (Robson and Cammidge); while the external secretion of the pancreas appears to be stimulated by the administration of adrenalin hypodermically (Pemberton and Sweet, 1910). Of clinical interest is the case reported by Lavenson (1908), in which symptoms supposed to be due to acute pancreatitis were found at autopsy to have been caused by hemorrhage into the adrenals. Löwi's test (1908) for pancreatic disease (mydriasis

from instillation of adrenalin into the eye), though by no means authoritative, is another evidence of the little understood relation of the pancreas and the adrenals.

In an interesting study of "The Theory and Treatment of Diabetes," von Noorden (1913) has endeavored to represent graphically the co-relationship of Pancreas, Liver, Central Nervous System and the Ductless Glands (Fig. 159). He says:

"The manufactory for sugar is the liver, and the liver cells constitute the working department.

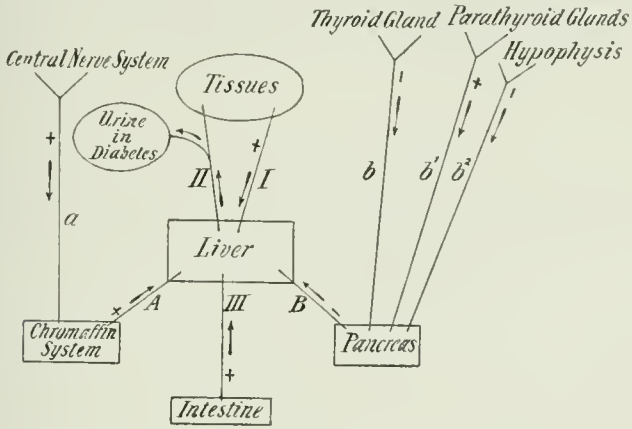


FIG. 159.—Diagram to Explain the Modern Theory of Diabetes. (*von Noorden.*)

"The important claims that *determine* the amount of the sugar production arrive from other organs and tissues, especially from the muscles (path I). The greater the consumption of sugar the stronger will be the claiming impulse, and an amount of sugar sufficient to meet the demand will pass into the blood (path II). This is normally the only influence which induces the liver to raise its output.

"However, along path III stimuli will also pass. This path represents the blood streaming into the liver from the intestinal wall, and carrying carbohydrates and the products of protein digestion. The quantity of these materials varies according to the type and composition of the food. So long as the manufacture of sugar is well under control the effect of this form of stimulus is not to induce a liberal outpuoring of sugar into the general blood stream, but to form glycogen. This glycogen is then stored until it is reconverted into sugar at the request of the tissues, and travels along path II to the muscles, etc.

"In order to maintain the excitability of the sugar manufactories at the proper intensity the two controlling factors come into play. The pancreas, which depresses the excitability, sends its secretion along path B, while the suprarenals, which increase the excitability, distribute their secretion along path A. This conjoined action serves to keep the process in equilibrium.

"Both these controlling glands are in turn influenced by other organs—the pancreas through the thyroid gland path (b), and the suprarenal through the nerve paths (a). The diagram shows still other influences (paths b^1 and b^2); their existence is most probable, but their origin is undetermined. They seem to be subordinate in importance and action."

General Diagnostic Considerations

Though disease of the pancreas is now recognized as not very rare, and the contention of Robson, that its diagnosis usually is possible is very generally accepted as true; it is nevertheless a fact, owing to the deep situation of the pancreas, and its close anatomical and physiological relations with surrounding organs, that such diagnosis frequently is difficult, and that pancreatic lesions often escape even acute observation.

The pancreas has both *digestive* and *metabolic* functions, the disturbance of which by disease causes more or less definite symptoms; and there are certain *physical signs* which usually may be elicited in cases of pancreatic disease.

Pancreatic Insufficiency.—If all diseases of the pancreas were attended by obstruction to the outflow of its secretions into the duodenum, the diagnosis could readily be made by means of the duodenal tube (Einhorn, Rehfuss) and the fractional analysis of the duodenal contents obtained by its means. This remains the best means of diagnosis whenever it is available and whenever there is complete insufficiency of the external secretion of the pancreas. Since the introduction of this method the elaborate methods (mentioned in our first edition), for determination of the presence of the pancreatic ferments in the feces, have lost most of their interest; and only a brief account of them is retained in the present volume.

Digestive Symptoms.—It is well known that the digestive functions of the pancreas may be partially assumed by other organs. Possibly if the pancreas could be removed piecemeal, destroying its functions one by one in very gradual stages, the loss of its secretions might be

entirely supplied by compensatory action of the salivary glands, the gastric and the intestinal juices. The amylolytic function of the pancreas is supplemented by the saliva; the tryptic function is the same as that of the pepsin of the gastric juice; and the intestinal juices and bacteria, aided by the bile, are capable of caring for a large part of the ingested fat. But when disease attacks the pancreas, the destruction of its physiological functions often takes place so suddenly that very pronounced and definite symptoms are produced. Chief among these are *steatorrhea*, and *azotorrhea;* others are *sialorrhea*, *dyspepsia* (a vague term), *emaciation*, *nausea*, *vomiting*, etc. The administration of pancreatic extract, pancreatin, etc., as a means of obviating these symptoms, was used as a diagnostic test by Salomon (1908) and others.

Steatorrhea.—An excess of fat in the feces was first recognized in 1820 by Kuntzmann as due to pancreatic disease.

The excess of the fat in the feces may be evident to the most casual observation, or may be demonstrable only by the microscope. In well-marked cases of steatorrhea the passages are bulky, of a silver, gray, or asbestos-like color; and the fat may float on the surface of the fluid mass like oil droplets or particles of butter. Such passages have been known occasionally to occur in health, after the ingestion of abnormal quantities of fatty food, as individuals differ in their ability to digest fats; and diminution of the secretion of bile, diarrhea, caseation of the mesenteric lymph-nodes, and other intestinal derangements, as well as pancreatic disease, may give rise to this symptom.

Azotorrhea.—The presence in the feces of undigested proteid material was first recognized as a symptom of pancreatic disease by Fles, in 1864. If gastric digestion be deficient, the meat fibres will not be separated from each other, and the trypsin of the pancreatic juice will be able to act only on the surface of the meat bundles.

Schmidt (1908) observed that a safer test than mere digestion of the meat fibres was the *destruction of the nuclei of the muscle cells*, since these are digested only by the pancreatic juice; his test consists in feeding to patients, suspected of having pancreatic disease, small cubes of beef enclosed in little silk bags; these bags are readily recovered from the feces, and their contents are examined microscopically to determine whether by penetration of pancreatic juice the muscle-cell nuclei have been destroyed.

Müller's test, to determine the presence of a proteolytic ferment (trypsin) in the feces, is described by Lyle (1910): after the administration of a test-meal, and suitable preparation of the fecal mass, small

drops of the latter are plated on a Petri dish of coagulated blood serum, and this is incubated; if a proteolytic ferment is present, small depressions are produced in the blood serum wherever the fecal drops have been placed.

Sahli's test (1898, 1902) for the efficiency of proteid digestion depends on the fact that gelatin capsules hardened in formol resist gastric digestion for twelve hours or more, but are rapidly digested by pancreatic juice. By administering such capsules filled with iodoform the fact of their disintegration can be proved by finding iodine in the urine or saliva; the reaction appears in health in from four to eight hours, and if there is no marked loss of gastric motility a delayed reaction indicates impairment of pancreatic function. The test is by no means accurate, but may sometimes aid in making a diagnosis.

Salomon's test (1908) depends on the fact noted in 1898 by Deucher, that lecithin is present in unusual quantities in the feces of patients with pancreatic disease. Salomon found that in patients with pancreatic disease on an egg diet from 0.4 to 1.2 gram of lecithin are excreted in the feces daily; whereas if the pancreas is normal, never more than 0.1 gram is excreted. He states that in uncomplicated cases of biliary obstruction Jürgensen found the quantity varied daily from 0.1 to 0.4 grams. Salomon also claims that patients with pancreatic disease who are fed on v. Noorden's oatmeal diet have typical "butter stools."

Carbohydrate Digestion.—Fedeli and Romanelli (1909) described a test for the determination of the functional activity of the pancreas, which they found was quite as accurate as Müller's test: their test is based upon the fact, proved by Roger and Simon, that saliva is inhibited by the gastric juice but reactivated by the pancreatic juice if in an alkaline medium. By mixing a certain portion of the patient's saliva with gastric juice (or HCl) and an indicator (carbohydrate), and then rendering the mixture alkaline, the presence of pancreatic ferment in the feces is shown by its reactivating effect on the saliva, which digests the carbohydrate indicator when feces and saliva are mixed.

The presence of **stercobilin in the feces** indicates that there is not complete obstruction to the discharge of bile into the intestines. Robson and Cammidge (1907) found that it is entirely absent or present in only very faint traces in cases of carcinoma of the head of the pancreas, where biliary obstruction usually is complete at the time these patients come under the surgeon's observation; whereas

in cases of obstructive jaundice due to other causes (chronic pancreatitis, common duct cholelithiasis) the obstruction is rarely absolute, and a distinct though often subnormal reaction for stercobilin may be obtained. As previously remarked, it is much simpler and very much more accurate, at the present day, to examine the patient's duodenal contents.

Other matters in the patient's stools may call attention to the pancreas as the seat of the disease. While there is nothing pathognomonic about *blood* or *pus* in the motions, yet the periodic discharge of *saliva-like fluid*, which has been noted in some cases of pancreatic cysts, or the passage of pancreatic calculi, could scarcely fail to arrest the attention of both patient and surgeon. Chiari's and Trafeyer's patients (1880) passed a *gangrenous pancreas* by rectum.

Sialorrhea Pancreatica.—Under this name is described an increased flow of saliva which has been observed in a few cases of pancreatic disease, and which is regarded by Robson and Cammidge as significant of efforts at compensation; in one of their patients troublesome salivation "ceased in a most striking manner within forty-eight hours after he had been operated on for chronic pancreatitis." Carnot (1908) referred to other cases recorded by Battersby, Ludolph, Holzmann, Caparelli, and Guidiceandrea.

Dyspepsia due to pancreatic disease is difficult to differentiate from those forms caused by biliary or gastric affections. Anorexia is more marked in gastric affections. Loss of weight in spite of ingestion of nearly normal amounts of food frequently characterizes pancreatic disease. Pain is less acute than in gastric dyspepsia, and attacks of colic much less usual than in gall-bladder disease.

Emaciation, as already noted, is often a prominent and highly suggestive symptom of disease of the pancreas. The loss of flesh is rapid, persistent, and very generally observed by both patient and physician.

Metabolic Symptoms. Glycosuria.—As was pointed out in the section on pancreatic diabetes, this condition is associated with change in the islands of Langerhans, and occurs only when their destruction is very widespread; moreover, pancreatic diabetes may be unaccompanied by any symptoms of impairment of the external secretion of the pancreas. Glycosuria, therefore, is not a frequent symptom of such pancreatic lesions as have interest for the surgeon; it usually indicates a very advanced lesion, which is most frequently chronic interstitial pancreatitis of the interlobular type. Interacinar pancreatitis, though producing glycosuria much sooner, is a much rarer affection.

Dextrose is the sugar usually found in the urine of pancreatic diabetes, but maltose and even pentose occasionally are present.

Alimentary Glycosuria.—Even though the diet of a healthy individual contains a great excess of sugar, it is only very exceptionally that glycosuria occurs during the process of digestion. But this alimentary glycosuria is much more apt to occur, and may be a constant phenomenon, if there is serious disease of the pancreas. It was found by Wille (1899), after whom the test frequently is named, that in about 65 per cent. of cases of alimentary glycosuria there is serious disease of the pancreas. Dextrose (fruit sugar) is better for the test than cane sugar: 100 grams of dextrose are given in a half pint of water on the fasting stomach, and the urine is examined two or three hours later.

Opie (1908) succeeded in one case in demonstrating the presence of a *fat-splitting ferment in the urine;* Hewlett (1904) had found a similar ferment in the urine of dogs for a period of from three to five days after experimental injuries of the pancreas.

Wohlgemuth and Noguchi (1912) found in experiments on dogs, after contusion of the pancreas, that *diastase was found in both blood and urine.* They think the test should be even more accurate in man than in the dog, and that diastase should appear promptly in every case of pancreatic lesion.

Physical Signs.—Inspection may show *emaciation,* which, if of rapid occurrence, in connection with digestive symptoms, is always suggestive of pancreatic disease. *Jaundice* is another physical sign very evident when it occurs, but by no means so frequent in pancreatic disease as the relation of biliary lesions to the latter might lead one to expect. The occurrence of jaundice depends upon the relation of the common bile-duct to the head of the pancreas; and Robson and Cammidge call attention to the coincidence of their figures with Helly's anatomical investigations: as was stated at page 621, Helly found the common duct embedded in pancreatic tissue in 62 per cent. of cases; and Robson and Cammidge found bile-pigments in the urine of 62 per cent. of those cases of chronic pancreatitis which were associated with cholelithiasis. The induration of the pancreas may be sufficient to obstruct the outflow of bile even if no gall-stones are present. Robson and Cammidge observed jaundice in 16 per cent. of such cases. Robson, Kehr, and others think that many cases of so-called "catarrhal jaundice" are due to pancreatitis of mild degree. Steadily increasing, painless jaundice, in time becoming almost black, with distention of the gall-bladder, is very characteristic of carcinoma of the head of the pancreas, or of the papilla of Vater.

Fat necrosis, first studied by Balser in 1882, may be seen when the abdomen has been opened; it is due, probably with very few exceptions, to pancreatic disease.[1] By fat necrosis is meant the result of the action of the steapsin of the pancreatic juice upon surrounding fat areas, resulting in the splitting of this fat into acids and glycerin. "Fatty acids," says Opie, "are deposited as needle-like crystals within the cell, which has lost its nucleus and is evidently necrotic, while the soluble glycerin is absorbed. Very soon the fatty acids unite with calcium, to form calcium salts; and within the cell outline which is still preserved are irregular, often globular masses, in which the presence of lime salts may be demonstrated by micro-chemical reactions." According to Ebner (1907) fat necrosis may occur (1) *directly by trauma*, permitting the access of pancreatic secretion to the tissues immediately surrounding; (2) *through the lymphatics*, as noted by Bryant, Böhm, and Gulecke; (3) *through the blood*, for although Tschepurowski found that the action of steapsin is inhibited in the circulating blood, yet Payr and Martina (1907) have found emboli composed of pancreatic cells. Fat necrosis which is found widely disseminated in the abdomen (omentum, mesentery) is more apt to be due to diffusion of steapsin through lymphatic spaces than to direct transperitoneal contact of the pancreatic juice; and certainly in the rarer instances of fat necrosis of the subperitoneal, subcutaneous, and pericardial fat, any direct transperitoneal access of the pancreatic secretion is out of the question. Bunge explained the occasional absence of fat necrosis in suppurative pancreatitis by rapid thrombosis of the lymph-stream.

The areas of fat necrosis vary in size from less than one to more than five millimetres, usually being visible as minute whitish specks or flakes, of dense rigid feel, often surrounded by a hemorrhagic zone, and not raised from the surface of the surrounding fat, a fact which aids in distinguishing them from miliary tubercles. It is not unlikely that very minute areas of fat necrosis often are overlooked. The whitish specks may be made more conspicuous, as Bender has shown, by the application of a half-saturated solution of copper acetate, which turns the affected area green, showing a fine contrast from the yellow of the normal surrounding fat.

Fat necrosis occurs in the acute lesions of the pancreas (infections and traumatisms) with much greater frequency than in chronic affections (interstitial pancreatitis, carcinoma), but it appears to have been definitely established that it is not itself caused by bacteria; these are a

[1] Richter (1910) noted its presence in a case of perforated duodenal ulcer and Marton (1919) in one of gastric perforation.

mere coincidence. In one case recorded by Hansemann (1889), areas of fat necrosis in the subcutaneous tissue were found to correspond with circumscribed hemorrhagic areas visible through the skin.

Hemorrhage.—Another characteristic of disease of the pancreas is a hemorrhagic tendency. On opening the abdomen in acute cases there may be found a bloody exudate bathing the entire abdomen; various discrete hematomata, sometimes widely distinct from the pancreas, may be encountered; and frequently the pancreas itself is the site of a massive hematoma, or diffuse petechial hemorrhages. It was pointed out by Körte, as long ago as 1894, that these hematomata were not scattered around by chance, but that their situation might be explained on anatomical grounds, owing to the disposition of the various "peritoneal leaves" or "fascias d' accolement" around the pancreas. Leriche and Arnaud (1909) claim that the sanguinolent effusion in the general peritoneal cavity is due to rupture of the gastro-colic omentum (either by trauma or by the digestive action of the pancreatic ferments), or leakage of pancreatic juice through the foramen of Winslow, or the escape somewhere of the bloody exudate arising originally in the pancreas. Turner (1920) has reported two cases of acute pancreatitis in which ecchymotic areas appeared in one case around the umbilicus, and in the other in the flank, from the direct action of the infiltrating pancretic secretions.

After operation on patients with pancreatic disease, especially infections and carcinoma, there is not seldom manifested a tendency to hemorrhage, not confined to the wound alone; oozing may occur from previously intact mucous membranes, and subcutaneous bleeding, causing disfiguring ecchymoses, may result from trifling causes. Truhart and Doberauer, according to Carnot (1908), accused the digestive action of the pancreatic trypsin on the neighboring tissues, opening venules and arterioles, as the cause of this hemorrhagic tendency; while poverty of the blood in lime salts, which are excreted in excess in cases of pancreatic disease, was thought by Robson and Cammidge to be a sufficient explanation.

Edema of the lower extremities, from pressure on the inferior vena cava, is a rare sign of pancreatic disease, as is *ascites* from pressure on the portal vein. The senior author has met with ascites in one case.

Palpation.—In emaciated patients, and especially in those with gastroptosis, the diseased pancreas often can be detected by palpation as an oblong tumor, lying transversely above the umbilicus, of firm consistency, and usually more cord-like than might be expected. Carcinoma of the head of the pancreas could be more often detected by

palpation, were it not obscured by a distended gall-bladder, one of the most characteristic physical signs of this lesion. In cases of subacute and suppurative pancreatitis it is almost always possible to detect a deeply-seated diffuse resistance in the epigastrium, which on opening the abdomen is found to have been caused by the pancreas, engorged with bloody and inflammatory exudate, or with pus. In cases of acute pancreatitis rigidity of the belly wall may prevent satisfactory palpation. Cysts of the pancreas almost always can be felt on palpation, but their differential diagnosis is more easily made by percussion, as will be presently described. Hydronephrosis, which in a few cases has resulted from pressure by the pancreas on the ureter, usually can be recognized by palpation (cases of Chvostek and of Boldt, quoted by Martina, 1907). On the other hand, in a case reported by Villard and Thévenet (1909), a calculous hydronephrosis simulated a tumor of the pancreas because it produced obstructive jaundice. Extravasations in cases of acute pancreatitis sometimes are more easily discovered by palpation in the left loin than in the epigastric region.

Percussion.—This is of most value in determining the relation of the stomach and colon to supposed cysts or tumors of the pancreas. By distention of these viscera with air, by means of a hand-bulb attached to stomach or rectal tube, it usually is possible to ascertain: (1) that a given tumor is retroperitoneal in origin; (2) that it presents (a) through the gastro-hepatic omentum, (b) through the gastro-colic omentum, or (c) below the transverse colon. By percussion, also, the size of the liver may be determined; when this is much enlarged, and is accompanied by a distended gall-bladder and deep jaundice, the diagnosis of carcinoma of the pancreas is very probable (Fig. 166).

Diagnosis.—In attempting to make a diagnosis in a case of suspected pancreatic lesion, the surgeon should consider: (1) The predisposing causes; (2) the clinical history; (3) symptoms; (4) physical examination; and (5) the results of the laboratory tests of duodenal contents, urine, feces, etc.

1. **Predisposing Causes.**—Under this heading usually are considered sex, age, race, etc.

Sex.—The male sex undoubtedly is more predisposed to pancreatic disease than the female; large statistics show that 65 per cent. of cases of pancreatic disease occur in men.

Age.—Most patients with disease of the pancreas are of middle or later life; in young men or women it is said to occur chiefly in hard drinkers, prematurely aged.

Race.—We are not aware that marked prevalence of pancre-

atic disease in any race has been observed; but in our own experience members of the Hebrew race seem to have been in the majority, as is also the case in disease of the biliary tract (page 479).

Habits.—It has long been taught that hard drinkers and high livers are exceptionally predisposed to pancreatic lesions. This has not been our own experience; we have found, however, that many of our patients with pancreatitis (acute or chronic) had arteriosclerosis from one cause or another.

Obesity is another factor which is looked upon by some as a predisposing factor of consequence.

Certain other diseases, of which arteriosclerosis has already been mentioned, undoubtedly greatly predispose to the development of pancreatic lesions. By far the most important of these is *biliary infection:* Quénu and Duval in 1905 computed that gall-stones were present in 50 per cent. of cases of pancreatitis; Egdahl (1901) said about 42 per cent. of cases of pancreatitis are associated with gall-stones; Kehr (1909) found evidence of chronic pancreatitis in sixty-nine (30 per cent.) of his last 220 operations for biliary disease; Mayo (1918) has found 90 per cent. of his cases of pancreatic disease caused by or coincident with cholelithiasis or other infection of the biliary tract. As Kehr points out, the higher percentage of cases of pancreatic disease reported in recent statistics is to be explained by more careful examination of the pancreas at operation, such investigation being indicated whenever the upper abdomen is explored. Of seventy-nine patients with chronic pancreatitis under the senior author's care in the Lankenau Hospital, seventy-two (91 per cent.) showed evidence of infection of the bile-passages; forty-two (53 per cent.) had gall-stones, and in thirty (38 per cent.) there was non-calculous inflammation. Statistics may also be given of the percentage of cases of gall-stone disease in which lesions of the pancreas are noted; thus Mayo found that only 359 patients (8.9 per cent.) in a series of 4000 cases of biliary disease had also noticeable lesions of the pancreas; Kehr found that in 24 per cent. of his patients with disease of the biliary tract palpable lesions of the pancreas were present. Perhaps it is just as well in this place to note that a positive diagnosis of chronic pancreatitis cannot always be made merely from palpation of the organ during an operation. Many a time such a diagnosis has been made, and when opportunity has offered later to test the diagnosis by the use of the microscope no lesions have been found. These probably are cases of pancreatic lymphangeitis. The influence of *general infections* in producing lesions of the pancreas has already been noted (page 614). Finally *injury* should be

remembered as a predisposing cause of considerable importance (page 689).

2. **History.**—A good clinical history of the patient is of the utmost importance. The previous existence of general infections, especially of typhoid fever, owing to its predilection for the biliary tract, the occurrence of jaundice, dyspepsia, colic, or "stomach cramps,"—any one of these factors, should bring to mind the possibility of pancreatic infection as a sequel.

3. **Symptoms.**—No matter how trivial at first glance, all symptoms should be cautiously weighed, as possibly having some bearing on the diagnosis of pancreatic disease. Thus dyspnea, which many will think can have no special diagnostic value, is dwelt upon by both Riedel (1903) and Musser (1908), as especially characteristic of pancreatic disease; the former states that when during an attack of supposed biliary colic dyspnea is a marked symptom the surgeon always should think of pancreatitis as a complication; Musser also notes it as a symptom characteristic of acute pancreatitis. Dyspnea is thought to be produced either by reflex nervous action through pressure on the solar plexus, or by means of toxic matters in the circulation. Collapse, which often is profound in cases of acute pancreatitis, is attributed to similar causes. Cyanosis is a not unusual accompaniment of collapse and dyspnea. Pain, in acute cases, is excruciating; in chronic pancreatitis and in cancer of the pancreas it usually is not very severe, unless perigastric adhesions exist, or unless neighboring nerve trunks are compressed. Pain is often referred to the left shoulder blade, or to the middle of the back between the shoulders. Epigastric pain may be increased after taking food, possibly, as suggested by Martina, because the physiological activity of the pancreas some hours after a meal causes the gland to swell up and distend its peritoneal capsule. A very severe pain may be caused by small, localized, intrapancreatic hemorrhages. The pulse rate may remain normal, in spite of the hemorrhagic nature of the pancreatic lesion, provided there is no escape of secretions of the pancreas from the confines of its capsule. Fever usually exists in cases of infection of the pancreas which have lasted for several days, though in cases of pancreatic apoplexy and other hyperacute lesions where collapse occurs the temperature may be subnormal.

4. **Physical Examination.**—This should be systematic and complete. *Inspection* shows the presence or absence of emaciation, jaundice, tumor, dyspnea, cyanosis, etc. *Palpation* may detect rigidity of the abdominal walls; tenderness to pressure in the epigas-

trium; a deeply placed sense of resistance or a well-defined tumor; fulness in the left flank; a distended gall-bladder, etc. *Percussion* enables the surgeon to determine the relation of the stomach and colon to any suspected pancreatic swelling; and to outline the liver and gall-bladder.

5. **Laboratory Tests.**—Many of these have already been mentioned. The most important are those for detection of the pancreatic ferments in the *duodenal contents.* For *examination of the feces:* 1. Excess of fat, and diminution in the proportion of split fat contained in the feces. 2. Presence of undigested proteid material, as determined by Schmidt's test (page 627). 3. Absence of proteolytic ferment as shown by the test of Fedeli and Romanelli (page 628). In the *examination of urine,* the most valuable test is: Alimentary glycosuria (Wille's test, page 630). The *pancreatic reaction in the urine,* described by Cammidge (1904), is no longer considered of special value. For detailed descriptions of these various tests the reader is referred to the original communications of the authors, and especially to the writings of R. Gaultier (1905) and of Terrier (1906).

Acute Pancreatitis

Under this category are included both catarrhal and parenchymatous inflammations. The acute catarrhal inflammations of the pancreas are neither so frequent nor so important in surgery as the parenchymatous inflammations. They are attended by moderate swelling of the head of the gland, which may compress the common duct and thus give rise to "catarrhal jaundice," which, as previously mentioned (page 630), often may be due to such a condition as this rather than to edema of the mucous membrane of the bile-duct or occlusion of its duodenal orifice. Suppurative catarrh of the pancreatic ducts has also been observed. But much more importance attaches to pancreatic lymphangeitis and to chronic catarrhal pancreatitis, which, as will be shown subsequently, are frequent forerunners of chronic interstitial pancreatitis; the pathogenesis, symptoms, and treatment of these conditions are discussed in connection with the latter subject (page 667).

The parenchymatous forms of acute pancreatitis were classified by Fitz (1889) as *hemorrhagic*, *suppurative*, and *gangrenous.* The differences are those of degree, rather than of kind; and while gangrenous pancreatitis nearly invariably, and suppurative pancreatitis frequently, is a sequel of the primary hemorrhagic change, either

form may arise independently, though the gangrenous rarely does so. The fullest account will be accorded the primary, hemorrhagic form.

Hemorrhagic Pancreatitis

The tendency to hemorrhage in disease of the pancreas has already been discussed; but it must not be thought that the use of the term hemorrhagic pancreatitis implies any form of inflammation peculiar to the pancreas alone. It is well known that in other structures a hemorrhagic form of inflammation is not seldom observed; and the more that is learned of pathological processes in general and of the special pathology of the pancreas in particular, the more evident does it become that the pancreas conforms to general pathological laws.

Pathogenesis.—Hemorrhagic pancreatitis has been produced experimentally (Carnot, 1908) by trauma, by injecting chemicals into the duct of Wirsung or directly into the parenchyma of the gland, and perhaps most interesting of all by injections of bile, gastric juice, and even normal pancreatic juice, trypsin, etc. In connection with this auto-digestive action of the pancreatic secretion, the important point to observe, as explained at page 36, is that the pancreatic juice is activated by a kinase with which it comes in contact only after leaving the pancreas. Now, it has been pointed out by Carnot that, under abnormal conditions, a kinase generated by leukocytes or even by bacteria, within the pancreas, can activate trypsinogen and convert it into trypsin, and that if this is produced within the pancreas it will have the power of digesting the surrounding proteid material. By a similar pathological law, the pepsin of the gastric juice is activated only by hydrochloric acid; and as it is held by some that auto-digestion of the stomach, in certain cases, may produce "round ulcer" in that organ, so from analogy it was suggested (Truhart, 1906) that trypsin may be the direct cause of intrapancreatic hemorrhages, producing veritable "round ulcers" of the pancreas (Desjardins, 1905); and Mayo (1918) speaks of these fulminating forms of pancreatitis as "perforations" of the pancreas. The pancreas, as is well known, is extremely susceptible to *post-mortem* auto-digestion; indeed it has been claimed by Chiari (1906) that this process begins in approximately 50 per cent. of cadavers within a few hours of death, or even during the agonal period; and he has expressed the opinion "that idiopathic hemorrhagic or gangrenous pancreatitis for the most part is nothing other than an intravital tryptic auto-digestion of the pancreas" (Williams and Busch, 1907). We believe a very reasonable theory to account for the pathogenesis of acute pancreatitis is that so ably

supported by Archibald (1919), namely *retrojection of bile* as a consequence of spasm of the sphincter of Oddi (see p. 619).

On the other hand, the toxic agent producing the hemorrhage has been thought to circulate in the blood or lymph streams, as in the toxemic theory of the origin of gastric ulcer (page 67), in typhoid fever, and in various other infections. The hemorrhagic lesions in the pancreas would then be produced by ulcerations commencing in the endothelial lining of the vascular channels. These theories of pathogenesis have not been verified as yet by pathological examinations; indeed the hemorrhages and inflammatory changes are often so widespread that little pancreatic tissue remains for histological study.

It is important to observe that fat necrosis and hemorrhage in cases of pancreatitis go hand in hand; the latter is said by Dieulafoy never to exist without the former, though fat necrosis has been observed in numerous cases without any evidence of pancreatic hemorrhage. We have observed cases, however, in which intrapancreatic hemorrhage existed, without evidences of fat necrosis elsewhere than in the gland itself. And as the area of fat necrosis may be widely disseminated, and not limited merely to the surface of the omentum, mesentery, etc., but situated deep within their substance; so, too, the hemorrhages of pancreatitis may be confined neither to the pancreas itself nor to a sanguineous peritoneal effusion, but often exist as distinct and separate hematomata, in the root of the mesentery, in the omentum, in the peripancreatic or perirenal fat, etc., etc. In other words both fat necrosis and hemorrhage may occur wherever the destructive pancreatic secretion is carried, whether its path is intraperitoneal, along the retroperitoneal tissues, through lymph-spaces, or through the blood stream; but this is theory, our knowledge at present not permitting of differentiation. Dieulafoy attempted to draw a sharp distinction between cases of hemorrhagic pancreatitis and cases of panceatico-peritoneal hemorrhage, the former being infectious in origin and the latter merely toxic, caused by the extravasation of pancreatic juice which produces both hemorrhages and fat necrosis in various parts of the abdominal cavity; it arises not infrequently in the course of a chronic pancreatitis. It may thus be understood that a "pancreatic apoplexy" so called was believed to arise without preceding infection or inflammation; probably such cases are not uncommon at the present day, and closer study of very early cases may more and more often show it to be true that infection follows the chemical destruction of the gland but does not precede or produce it (p. 620).

Clinical Etiology. *Sex.*—The disease is more frequent in men than in women; large statistics show that about 65 per cent. of cases occur in men.

Age.—Most patients are of middle or later life, though cases are not unknown in young adults. In young persons it is said usually to occur in hard drinkers, who have developed arteriosclerosis before the usual time of life. Brewitt (1908) and Körte (1911) have each of them operated successfully on a patient of sixteen years with acute hemorrhagic pancreatitis.

Obesity usually is considered an important predisposing cause: among eighty-three cases of acute pancreatitis, collected by Williams and Busch (1907), obesity was distinctly noted in fifty, and in many others the amount of adipose tissue was not mentioned at all.

Previous digestive disturbances have almost always existed; they may consist merely of gastro-duodenitis or of more serious affections. Archibald suggests the long abstinences from food which characterize chronic alcoholics and other patients with disturbances of the upper digestive tract as a factor tending to keep the sphincter of Oddi closed, thus predisposing to the retrojection of bile into the pancreas (p. 619).

Cholelithiasis, however, is not so frequent an accompaniment of acute as it is of chronic pancreatitis. Kehr (1901) observed acute pancreatitis in only 1 per cent. of his operations for gall-stone disease. The statistics collected by Quénu and Duval (1905) showed that, of cases of pancreatitis associated with gall-stone disease, 60 per cent. were cases of chronic pancreatitis, and 40 per cent. of acute pancreatitis (23 per cent. were cases of gangrenous or suppurative pancreatitis, and only 17 per cent. were cases of hemorrhagic pancreatitis). These figures agree very closely with those reported by other observers: Nötzel (1908) reported nine cases of acute pancreatitis, three of which were associated with gall-stones; of the eighty-three cases of acute pancreatitis collected by Williams and Busch (1907) thirty-three (40 per cent.) were associated with gall-stones; and Egdahl (1907) found that gall-stones were present in forty-four out of 105 cases of acute pancreatitis. Opie (1908) collected forty-three cases in which gall-stones and acute pancreatitis were associated, in nine of which a calculus had lodged near the termination of the bile-duct and may have permitted retrojection of bile into the duct of Wirsung (page 618). It was suggested by Williams and Busch that the passage of a gall-stone into the duodenum may so dilate the intestinal orifice of the common bile-duct as to facilitate regurgitation of duodenal contents into the pancreatic duct. Numerous reports indicate that acute

pancreatitis occurs by no means infrequently in a gland already the seat of chronic inflammation.

Typhoid Fever.—Musser (1908) observed acute hemorrhagic pancreatitis as a fatal complication of typhoid fever.

Trauma.—It is generally acknowledged that trauma may be a cause of hemorrhagic pancreatitis, the hemorrhage causing a place of lessened resistance and predisposing the gland to infection; moreover, the structure of the pancreas is such that very insignificant trauma may result in serious intraperitoneal hemorrhages.

Morbid Anatomy.—On opening the abdomen early in the course of a case of acute pancreatitis there may be found no exudate, and nothing to indicate disease of the pancreas, not even scattered areas of fat necrosis, until the pancreas itself is brought to view when it will be found pinkish in color, tense beneath its capsule, and almost ready to perforate. At a later stage, however, perhaps after the lapse of only a few hours, though sometimes not for several days, there usually is found a sero-purulent exudate, sometimes blood-stained or even grumous in character. In nearly all cases areas of fat necrosis are found in the omentum, mesentary or peri-pancreatic fat. This exudate should not be interpreted as a direct extension from the diseased pancreas in all cases; it is the evidence that the general peritoneal cavity, and especially the omentum, is reacting to the pancreatic infection just as it does to infections arising in the gall-bladder, the appendix, the Fallopian tubes, etc. In cases where the exudate is blood-stained, however, and especially where there is extensive fat necrosis, it is probable, as previously noted (page 631), that the proteolytic and steatolytic ferments of the pancreas have escaped from their normal habitat, by way of the lymphatic spaces or possibly transperitoneally. The lesser even before the greater peritoneal cavity may be invaded by the exudate which early in the course of pancreatitis is sero-purulent; later it becomes bloody, grumous, even chocolate colored; rarely it is frankly purulent. Hematomata may be observed in the root of the mesentery, around the pancreas, and in the peri-renal fat. The pancreas itself, as already mentioned, in the very early stages of the disease, may present no very noteworthy macroscopic changes except that it may be enlarged; but very soon it becomes infiltrated with blood, which is conspicuous because in isolated spots separated by yellowish white areas of normal pancreatic tissue. In the course of a few days, if death does not occur sooner, the pancreas may be converted into a reddish-black mass of necrotic fat and blood clots (Plate IX). The disorganization of the gland usually is so extensive that little of value can be learned from a

Slough of Pancreas Discharged through Drainage Tract in Left Loin, in a Case of Acute Pancreatitis. Recovery. Death 6 Years later from Diabetes. *Lankenau Hospital.*

Face p. 640

microscopical study. The hemorrhages are interstitial, rarely invading the ducts.

If the patient lives, the lesions of gangrenous pancreatitis may be observed after the lapse of ten days or two weeks. In this stage a large portion of the pancreas, usually the body or tail, may be found almost completely detached from the surrounding tissues, lying as a slough in the retroperitoneal fat. The evidences of general peritoneal infection are now very slight, but the entire lesser peritoneal cavity may be converted into an abscess containing foul-smelling, purulent, chocolate-colored exudate, with pieces of necrotic pancreas floating around loose in the fluid. In rare instances the stomach, jejunum or transverse colon may be perforated. In a case recorded by Chiari most of the pancreas (identified by Rokitansky) was passed from the rectum as a slough; this was also the case in a patient of Trafeyer. In a patient under the care of the senior author nearly the entire pancreas was discharged as a slough through a lumbar incision made for drainage (Plate IX). This case has been reported in full by Jurist (1909). The patient continued in good health for about six years, but eventually died with diabetes.

Lesions of the biliary tract, a common accompaniment of acute pancreatitis, may also be observed on inspection of the abdomen at operation or *post-mortem*. They require no particular description in this place, but it is perhaps well to note that fat necrosis has been observed in a few cases apparently as the result of leakage of the pancreatic juice through a perforation or rupture of the biliary apparatus, stomach or duodenum (Richter, 1910; Marton, 1919), the pancreas itself not being diseased.

Symptoms.—There is little doubt that as experience accumulates, it will be possible to recognize milder attacks of acute pancreatitis than the fulminating cases which hitherto almost exclusively have occupied the attention of surgeons. Prodromal symptoms, in the nature of digestive disturbances, stomach or gall-stone cramps, etc., are said to exist in 70 per cent. of cases; but often the acute symptoms arise so suddenly, and are of such an overwhelming nature, that the patient can give no detailed history of his previous condition, and such prodromal symptoms are discovered only by inquiries from the patient after recovery from operation, or from his friends after his death. The disease usually runs its course in from five to eight days, death occurring within a week in the great majority of cases without operation. In a few cases, however, if no operation is done, the symptoms abate, and when about the tenth day the hemorrhagic or purulent effusion has

become localized, physical examination may enable a diagnosis of suppurative pancreatitis to be made (page 654).

The attack is characterized by both *abdominal* and *constitutional* symptoms. Of the first, *pain* and *vomiting* are the most important, and of the latter, *collapse*.

Pain.—This occurs suddenly in the epigastric region, and may be so severe as to cause faintness or collapse. It is a colicky pain at first, probably from sudden overdistention of the gland as the result of intrapancreatic hemorrhage. Sudden death may arise by inhibition of the heart. A rapid, but not sudden, death is more apt to be caused by toxemia. The pain resembles in its colicky nature that due to intestinal obstruction, but often it is extremely severe at its first onset, whereas the pain of obstruction frequently begins with mere twinges and becomes severe only after the lapse of hours. The pain of pancreatitis does not shift its position, but remains constantly epigastric, usually more to the left than the right of the median line. Pain may also be felt in the dorsal region, usually to the left of the spine, or in the left shoulder blade. It seems not unlikely in those cases attended by extremely severe pain, which cannot be relieved by morphin and which impels the patient to rise from the bed and walk around the room and frequently change his position, and which are not attended by marked collapse—that in these cases the hemorrhagic exudate is still confined by the capsule of the pancreas, not having broken through into the general peritoneal cavity; and that it is the latter event which brings on collapse. We have observed a few such cases where this state of affairs was demonstrated at very early operation. In such patients the pulse may remain full and strong and of normal rate. This may serve to differentiate the condition from intestinal obstruction in which the pulse rate usually rises rapidly even before the onset of peritonitis and fever.

Vomiting is an early and important symptom. It follows closely after the initial pain and is repeated so frequently as to resemble that due to intestinal obstruction; but in the latter condition the vomiting is projectile, there is little or no nausea and retching, and the vomitus soon becomes bile-stained and then fecal. In acute pancreatitis, on the other hand, the vomitus is never fecal, and frequently (when the bile-duct is obstructed) it is not even bile-stained; there usually is considerable nausea and retching, the gastric and duodenal contents being brought up only with difficulty. *Hiccough* is a frequent symptom, is often repeated, and very persistent.

Jaundice is of rather frequent occurrence in acute pancreatitis

either from primary calculous obstruction, or secondarily from compression of the bile-duct by the diseased pancreas.

Emaciation.—This, which is extremely rapid, is very characteristic of pancreatitis in all its forms.

Collapse.—This is not seen, as a rule, until perforation into the general peritoneal cavity occurs. Then the extremities are cold, the face and hands are covered with cold sweat, the nose looks pinched; there is mental hebetude; sometimes great restlessness and thirst; delirium is rare, except in later stages. The symptoms of collapse probably are due to the absorption of toxins of the broken-down pancreatic tissue (Egdahl, 1907); mechanical irritation of the peritoneum and stimulation of the celiac plexus are also secondary causes.

Pulse.—In the presence of collapse this is rapid, feeble, and often indicates the hemorrhagic nature of the abdominal disease; but as already noted, so long as the toxic extravasation is confined within the capsule of the pancreas, the excruciating pain has dominated the picture and collapse has been absent, and the pulse has been slower than normal and quite strong (Elliot, 1910).

Temperature.—This may be subnormal and is seldom high, early in the disease. When the collapse passes off, and under the influence of the peritonitis, it may be high, and in cases of abscess of the pancreas may assume a hectic type.

Dyspnea.—This, which perhaps is more accurately described as hypernea, has been noted by several keen observers as a symptom characteristic of acute pancreatitis. It is probably due in part to mechanical interference by the engorged pancreas with the action of the diaphragm; in part to the severe pain, and in part to the toxemia as in the parallel case of uremic dyspnea.

Convulsions, followed by *coma* and *death*, were the chief symptoms noted in a case reported by Tomaschny: as the patient (an old woman with senile dementia) had never had epilepsy and as the kidneys were normal and there was no diabetes, Tomaschny concluded that the convulsions were either reflex, from pressure on the solar plexus, or toxemic in origin; on the under surface of the dura mater there were some small yellow spots perhaps areas of fat necrosis; this was also present in the peripancreatic fat.

Physical Signs.—Thorough physical examination usually is impossible at the first onset of the disease, owing to the extreme degree of abdominal pain and tenderness. *Inspection* shows a slightly distended epigastrium, with thoracic breathing; cyanosis is frequent and livid splotches on the surface of the abdomen have been noted

several times (p. 632). On *palpation*, the muscular rigidity is not found to be very marked; in a few cases gentle but persistent examination of the epigastrium with the warm flat hand has enabled the surgeon to detect that the swelling was diffuse, and not simply dependent on a distended stomach or colon. Gentle palpation in the left costo-iliac space may discover an abnormal fullness, as well as exquisite tenderness, from the pancreatic extravasation in the retro-peritoneal tissues. *Percussion* may demonstrate an area of dullness in the region of the pancreas, between the ensiform process and the umbilicus.

Clinical Course.—After the extremely sudden onset of an attack of *acute pancreatitis*, characterized by violent epigastric pain, repeated vomiting, and collapse; the symptoms of peritonitis supervene, and are the dominant feature of the case during the second and third days of the disease. After this time, unless death occurs, the symptoms generally grow less severe, and the physical signs of *subacute pancreatitis* (gangrenous or suppurative stage) arise. The patient continues to be gravely ill, though suffering less intensely than at first; the stomach is unretentive, though vomiting usually is absent if entire abstinence from mouth feeding is persisted in, as it should be; emaciation is rapid; slight jaundice frequently is present; the pulse is weak and running; the temperature elevated (100° to 102°F.) and sometimes assumes a hectic type. In the epigastrium an indistinctly outlined and deep-lying tumor usually can be detected by palpation, and the rest of the abdomen may be no longer painful. The patient will now die of exhaustion, sepsis or secondary peritonitis from rupture of the pancreatic abscess, unless promptly relieved by operation.

Differential Diagnosis.—Unless a surgeon has seen previously two or three cases of acute pancreatitis, or unless he keeps the condition constantly in mind, it is seldom that a correct diagnosis is made before opening the abdomen; and it is generally admitted that any attempt to distinguish between the various forms of acute pancreatitis (hemorrhagic, suppurative, gangrenous), in the present state of our knowledge, is utterly futile.

The conditions for which acute pancreatitis is most often mistaken are biliary colic; acute intestinal obstruction; perforation of the stomach, duodenum or gall-bladder; and appendicitis.

Biliary colic presents many of the symptoms of acute pancreatitis, and a distinction may be very difficult. In acute pancreatitis, however, there usually is not a history of recurrent attacks; it is more apt to follow overeating than is biliary colic; and the pain though subject to exacerbations, as in biliary colic, scarcely ever is entirely

absent between these exacerbations; the pain, moreover, is very much more intense in the pancreatic affection—indeed it is seldom relieved by the morphin which may be given in large and repeated doses under the impression that the condition really is biliary colic. In pancreatitis there is collapse, cyanosis, and a sense of impending death, which seldom are noted in cases of biliary colic.

Intestinal Obstruction.—The difference in the character of the onset and in the nature of the pain have been mentioned already (page 642): in addition, there frequently is a history, in cases of acute intestinal obstruction, of a previous attack of peritonitis which might have left behind crippling adhesions, etc. The collapse is not so great as in pancreatitis, the temperature is not elevated, and the onset of peritonitis, with fever, wiry pulse, and distended abdomen is more delayed. The vomiting is projectile, with little or no nausea or retching, and rapidly becomes fecal in the case of obstruction; whereas in pancreatitis the nausea is marked, the gastric and duodenal contents are brought up only with effort, and though the vomiting may be often repeated (every ten to fifteen minutes), fecal vomiting scarcely ever is observed. A slight icteric tinge of the sclera is present sometimes in pancreatitis, but is very rare in intestinal obstruction; persistent absence of bile from the vomitus speaks in favor of pancreatitis. In both affections there nearly always is absolute constipation, and in both an evacuation of the lower bowel sometimes may be secured by the use of enemata even after the abdominal symptoms are well advanced. In most cases of pancreatitis, however, the *peritoneal* overshadow the *intestinal* symptoms, while the reverse is the case in the early hours of obstruction. Active peristalsis will be heard in obstruction, but will be absent in pancreatitis. Physical examination is more apt to be negative in cases of pancreatitis than in cases of intestinal obstruction, since here the existence of a tumor (intussusception, volvulus) sometimes may be demonstrated. Dyspnea and cyanosis, if present, point to pancreatic disease. In addition to all the above differential points, it must be remembered that intestinal obstruction is more common in the young (intussusception, Meckel's diverticulum, appendicitis) and in the old (carcinoma, strangulated hernia, volvulus), while pancreatitis is most frequent in males of later middle life.

Perforation of the Stomach, Duodenum or Gall-bladder.—In these conditions it is possible in the vast majority of cases to obtain a history of gastric or biliary symptoms extending over a number of years. In cases of perforation of the gall-bladder the patient almost invariably

will have been confined to his bed for some days at least with upper abdominal symptoms, and frequently the distended gall-bladder can be recognized before perforation occurs. The onset of the attack in cases of perforation of a hollow viscus, though often quite as sudden as in acute pancreatitis, and though attended by very severe pain, is yet unattended by the marked collapse which is characteristic of the latter condition, and the vomiting seldom is repeated. As operation is demanded even at an earlier period in cases of perforation than in acute pancreatitis the differential diagnosis is of more academic than practical importance. (See also page 91.)

Appendicitis.—While the initial pain in appendicitis is umbilical and colicky, as is often the case in acute pancreatitis; yet it is not so severe, collapse is rare, and vomiting is not repeated. The pain of appendicitis localizes itself in a few hours to the right iliac region, and any mass which forms will be here or in the pelvis; while a palpable mass forming as the acute symptoms of pancreatitis subside will be in the epigastric region, slightly to the left of the median line, or in the left loin. The age of the patients is different, and a history of previous attacks of appendicitis may throw further light on the diagnosis.

Gynecological affections, such as ovarian tumors with twisted pedicle, rupture of a tubal pregnancy, etc., usually may be excluded by attention to the history and a careful pelvic examination.

Poisoning.—Leriche and Arnaud (1909) suggest that the cases of hyperacute pancreatitis ("pancreatic apoplexy"), followed by death in a few hours in a state of collapse, might be mistaken for poisoning by drugs and they recommend that pancreatitis be remembered as a cause of sudden death in cases having a medico-legal aspect.

Prognosis.—Acute pancreatitis is now regarded as a surgical disease; this is conceded by most physicians, and there are good reasons why it should be so. Under purely medical treatment the vast majority of patients with acute pancreatitis died. In 1908 Dreesmann collected case records from the past ten years, which showed that of thirty-six cases of acute pancreatitis treated without operation, only four recovered; a mortality of 88 per cent.; while of 118 cases subjected to operation fifty-three recovered, a mortality of only 55 per cent. The statistics collected by Ebner (1907) may be added: of twenty patients treated without operation, eighteen died, a mortality of 90 per cent.; of thirty-six patients subjected to operation, nineteen died, a mortality of 47.2 per cent.; and while, as Dreesmann suggests, such statistics as these no doubt give an unduly favorable record for the operative cases, since successful are more apt to be reported than

unsuccessful operations, yet the difference between the medical and surgical results is so extreme that little further argument is needed.

With this limitation in mind, it is interesting to see the gradual improvement in operative results which is evidenced in the following statistics.

In 1906 Lenormant and Lecène found six recoveries among thirty-six operations (mortality 83.3 per cent.).

In 1909, Leriche and Arnaud found (since 1906) thirteen recoveries among thirty-nine operations (mortality 66.6 per cent.).

In 1911, Körte found among 118 isolated operations reported since 1905, seventy-three recoveries and forty-five deaths (mortality 38 per cent.).

In 1917 Linder reported a series of operations, collected within the previous three years. Up to 1915, there were 16 operations with a mortality of 62.5 per cent. In the second series, since 1915, comprising 15 operations, the correct diagnosis was made in 75 per cent. of the cases, and the mortality was only 13.6 per cent.

Bloodgood (1919) reports a series of 11 operations, with 3 deaths (27.2 per cent.).

It has been argued that patients who are finally subjected to operation sometimes give a history of having passed through similar attacks before, recovering without operation; but it is even more difficult to diagnose an attack of acute pancreatitis from vague descriptions given by the patient of a previous illness than it is when we see him during the attack; and it seems not impossible that some of these acute attacks from which the patients are alleged to have recovered have been attacks of biliary disease. Yet it is true that some of the operations followed by recovery (Halsted, Dick) consisted in little more than opening the belly and sewing it up again; but one or two cases are not enough to controvert the dictum that without operation the prognosis is shockingly bad.

The first recovery after operation with drainage is credited to Hahn (1900).

As to the time at which operation shall be undertaken, surgeons are gradually becoming unanimous in their view that immediate intervention offers most chances of success. All those whose experience has been greatest in the treatment of acute pancreatitis, urge immediate operation; and even Körte, who at first (1898) advised waiting until the subacute stage on the ground that no operation could be of benefit in patients so acutely ill, revised his teaching in 1907, and then asserted his opinion that operation as soon as possible after the onset of symp-

toms affords the best chance of cure. And as we learn more of the pathology of the affection, this seems the only reasonable ground to take: the conditions in acute pancreatitis are in many respects similar to those present in traumatic lesions of this region—escape of pancreatic juice into the peri-pancreatic tissues and the general peritoneal cavity, and hemorrhage. If the case were traumatic in origin, no one would counsel delay in opening the abdomen and attempting to evacuate the toxic fluid (absorption of which causes the collapse, etc.) and to check the bleeding by tampon or suture. As a matter of fact, however, very few operations have been done within a few hours of the onset of the attack; usually the patient does not come under surgical care until the second or third day of the disease,[1] and we believe that under such circumstances, as in other cases of diffuse peritonitis, it is safer sometimes to encourage localization of the process before instituting drainage. But it should be distinctly understood that by urging postponement of operation at this stage we do not mean to leave the patient alone until he is nearly moribund from sepsis: sometimes it may be possible to wait until a well-localized tumefaction indicates the presence of an abscess or of gangrene; but whenever a case is seen before symptoms of diffuse peritonitis arise, the surgeon should lose no time in opening the abdomen to evacuate the extravasated toxic fluid. In general, we believe that immediate operation should be done, unless such a course manifestly would hasten death; under such circumstances, and when the patient is first seen when diffuse peritonitis is well advanced, it will be safer to postpone operation, relying on the "Ochsner treatment" to encourage localization of the process.

Acute Pancreatitis. Operation in the Subsiding Stage; Recovery

A Polish woman, 24 years of age was admitted to the German Hospital Nov. 5, 1916. She had been perfectly well until two days before admission, when she was seized with the most acute abdominal pain, followed by vomiting.

On admission the abdomen was greatly distended, rigid and universally tender; the tenderness was more marked 5 cm. above the umbilicus and to the right of the midline. A tentative diagnosis of acute appendicitis with peritonitis was made, and the patient was treated by anatomical and physiological test, being placed setting up in bed with ice to the abdomen, and nothing by mouth. At the end of three days peristalsis was audible but no flatus had been passed. The abdomen was becoming relaxed below but was still rigid above the umbilicus. Two days later there was an area of dulness demonstrable in the upper left quadrant of the abdomen. Cystoscopy and catheterization of the ureters

[1] Thus in Hahn's case (1900), already alluded to, operation was not undertaken until three days after the onset of the disease; Bunge's patient (1903) quoted far and wide as an incontrovertible proof of the value of immediate operation, was not operated on until sixty hours after the onset of the attack.

were negative. X-ray examination showed merely a diffuse shadow in the upper left quadrant.

The diagnosis was now revised to *pancreatitis with effusion.*

Operation (5 days after admission, one week after onset of illness), by Dr. Deaver, demonstrated widespread fat-necrosis. The stomach was pushed upward and there was bulging of the thickened gastro-colic omentum, where a fluctuating area was found. Incision evacuated a large amount of pus and bloody fluid from the lesser peritoneal cavity. Drainage was provided by two wide rubber tubes, coffer-dammed by three gauze packs each 30 cm. by 100 cm. in size.

Uneventful recovery; 3½ years later she was reported to be in good health.

It is almost impossible to secure figures which will give any clear indication of the truth in this matter, for the case reports as a rule do not give the number of hours elapsed between onset of the attack and operation, nor do they clearly define the stage of disease (hemorrhagic or suppurative) in which operation is undertaken; and even if such figures were compiled, from scattered case reports, they could not convey the real truth, because successful operations are always reported sooner than unsuccessful. Nor would the patients who had died without operation be included in such statistics. Körte (1912) attempted to show the results obtained by those who have reported more than one operation (presumably their entire experience in each instance); he collected in this manner reports of 103 operations: among these, forty-one patients recovered, and sixty-two died, a mortality of 56.9 per cent. Körte's own experience (embracing ten cases of his colleague Brentano) includes forty-four cases of acute pancreatitis, in the period from 1890 to 1910. Only cases where the diagnosis was proved at operation or at autopsy are counted. In six cases no operation was done, and all the patients died. In thirty-eight cases operation was done: in four cases the gall-bladder was drained and nothing was done to the pancreas; all the patients died. In thirty-four cases the usual operation on the pancreas was done, with eighteen recoveries and sixteen deaths (47 per cent. mortality).

KÖRTE'S OPERATIONS

Acute Pancreatitis	Total	Recovered	Died	Mortality per cent.
In the first week of the disease	12	8	4	33.3
In the second week of the disease	4	3	1	25.0
In the third week of the disease	7	4	3	43.0
In the fourth week of the disease	7	3	4	57.0
From the fifth to the seventh week	4	0	4	100.0
	34	18	16	47.0

There are few, if any, surgeons who have had so extensive an experience with this disease as has Körte. In the table published in the first edition of this work, we assembled the statistics of those surgeons whose experience most nearly approached his. This comprised a total of 193 cases with 119 deaths, a mortality of 61 per cent.; 32 of these patients were treated without operation, with 8 recoveries and 24 deaths, a mortality of 75 per cent.; while 161 patients were subjected to operation, with 67 recoveries and 94 deaths, a mortality of 58.3 per cent.

As is the case in every other department of surgery, the mortality of operative treatment begins to decrease as the disease in question becomes better recognized, and particularly as the individual surgeon's experience increases and as his judgment and his technical skill improves. So that at the present day the death rate is considerably less than indicated by the figures just quoted.

In the first edition of this work the senior author recorded 11 operations for acute pancreatitis, with 5 deaths a mortality of 45 per cent. Since 1912 he has operated on 24 patients, with only 9 deaths (37.5 per cent.).

OPERATIONS FOR ACUTE PANCREATITIS (1912–1920)
(Lankenau Hospital)

Operation	Associated lesions	Cases	Deaths
Cholecystostomy	No other lesion	3	2
	Calculus	3	0
	Pancreatic abscess, calculus	1	0
	Cirrhosis liver	1	1
Cholecystostomy and pancreatostomy	No other lesion	2	1
Cholecystectomy and choledochostomy	No other lesion	1	0
	Calculus	2	0
	Pancreatic abscess	1	1
	Pancreatic abscess, calculus	1	1
Cholecystectomy and pancreatostomy	Calculus	6	1
Pancreatostomy	No other lesion	3	2
		24	9 = 37.5% mortality

Treatment.—When operation is undertaken, a certain definite technique should be followed. In cases where the diagnosis is un-

certain, the surgeon may make his first incision in the hypogastric region; but if he is an acute clinician it usually will be possible for him to determine before beginning whether the seat of disease is in the lower or upper abdomen. In any case, however, pancreatic disease is to be suspected so soon as the abdomen is opened if there is a bloody turbid exudate, or if there is fat necrosis; marked distention of the transverse colon, without apparent obstruction, is another valuable sign. If only an exploratory hypogastric incision has been made (as is best in cases of uncertain diagnosis), it should be temporarily plugged with gauze; an incision is then made in the epigastrium, through the left or right rectus. The pancreatic lesion may now be self-evident; and the stomach may be displaced by the swollen gland simply forward, or upward or downward; rarely the colon will be displaced upward, and the pancreas prove most accessible through the transverse meso-colon. When a choice is possible, the surgeon should aim to expose the pancreas through the gastro-colic omentum, dividing this structure between the gastro-epiploic vessels and the colon, as described at page 295 (Fig. 160); but if the pancreas bulges through the gastro-hepatic omentum or through the transverse meso-colon, it should be approached by that route.

FIG. 160.—Wide Opening of the Gastrocolic Omentum to Explore the Pancreas.

Having thus exposed the pancreas itself, many surgeons have been content to conclude their intervention at this point by tamponing the lesser peritoneal cavity, without opening the capsule of the pancreas. Nötzel (1908), Stieda (1910), and others who recommend this plan contend that incision of the swollen hemorrhagic pancreas may lead to uncontrollable hemorrhage, and will give exit to exceedingly toxic substances which will be more rapidly absorbed by the peritoneum, and thus more quickly destroy the patient, than if they were still confined to the substance of the inflamed gland.

Direct incision of the pancreas was first proposed in 1893 by Nimier, who suggested controlling the hemorrhage by the actual cautery or by gauze packing. It appears to have been put into execution first by C. B. Porter in February, 1903, closely followed (December, 1903) by Muspratt, both patients recovering.

As far as our own experience goes, and from what can be gathered from case reports of other surgeons, there is no object in incising the pancreas unless it appears distended, or unless there is a hematoma present. In several cases operated on in the hemorrhagic stage (Bunge, Hahn, Coenen, Nötzel) the evidences of intra-pancreatic damage have been comparatively slight, and recovery has followed mere tamponade of the lesser peritoneal cavity. In Coenen's patient (Case 1) there was no exudate at the time of operation, but seven days after operation there occurred a profuse discharge of pancreatic secretion from the wound, with sloughs of the pancreas; and in Nötzel's patient (Case 3) no cause for the peritonitis was found at the time of operation, so the wound was tamponed, but reopened during the subacute stage when the pancreatic exudate was evacuated. The cases recorded by Bunge and Hahn are similar.

On the other hand we do not think such great fear of systemic poisoning need be felt if the distended pancreatic capsule is incised to give exit to dammed-up secretions or blood clots; indeed it seems not improbable that there is less danger of absorption from the serous surface of the peritoneum than from the retroperitoneal cellular tissues. In every case, of course, the pancreas should be opened only after carefully protecting the general peritoneal cavity by a coffer-dam of gauze; and if there is any localized collection of fluid, this may well be evacuated first by an aspirator, still further to decrease the chance of soiling the peritoneum, the abscess or hematoma being formally incised only after its contents have been withdrawn.

Bircher (1910) successfully excised the entire tail of the pancreas which was the seat of a hematoma on the third day after onset of symptoms; and he urges excision whenever possible, as the removal of the focus of disease, as of a gangrenous appendix, should accelerate the cure.

Another question in dispute is whether or not any treatment of biliary complications should be undertaken when the abdomen is opened in a case of acute pancreatitis. This was urged as a measure of routine by Ebner (1907), but the proper course, it seems to us, depends upon the state of the biliary tract and the condition of the patient. Should the latter warrent such interference, we see no good reason for postponing drainage of the gall-bladder if this is easily accessible and is the seat of acute cholecystitis, or contains calculi; if on the other hand the gall-bladder is buried in adhesions, if there is doubt as to its being acutely diseased, or if for any reason the calculi present are particularly inaccessible, it no doubt would be proper to

postpone to a more opportune time any indicated operative treatment of the biliary tract, even if the condition of the patient were reasonably good. Rarely we believe, will the condition of the patient justify cholecystectomy or any procedure more radical than drainage of the gall-bladder; though in the cases in which we have removed the gall-bladder the mortality has been lowest of all. When because of the patient's condition it is necessary to postpone surgical treatment of biliary lesions, the surgeon should not forget that a diseased biliary tract will require treatment sooner or later. It is better to do two operations on a living patient, than to do one thorough operation and have a dead patient.

Archibald (1919) suggests the propriety of incising the papilla of Vater by duodenotomy, to prevent further damming up of bile; but we belive that in most cases choledochotomy with passage of a sound down the duct into the duodenum will prove safer.

Many surgeons still urge free irrigation of the peritoneal cavity with saline solution; they claim that not only is it important to secure evacuation of all the exudate, but that owing to its toxic nature it is extremely desirable to dilute it. As cultures from the bloody exudate have in a large number of cases shown it to be sterile, and as sufficient evacuation is secured through absorption by the gauze packs used during the operation, it seems to us quite unnecessary to irrigate the abdominal cavity; the practice probably is not as undesirable as in cases of appendicular peritonitis, but that is the most that can be said in its favor. In all cases where much effusion is found in the general peritoneal cavity, the pelvis should be drained through a suprapubic incision.

We may sum up the technique of operation for acute pancreatitis as follows: through an epigastric incision isolate the pancreatic region by gauze packs; if a collection of fluid exists, evacuate it by aspiration; expose the pancreas preferably through the gastro-colic omentum, and if it presents no gross lesions do not incise it, but merely tampon the lesser peritoneal cavity; if there is a hematoma or abscess in the pancreas, incise its capsule, and with a blunt instrument carry the incision into the substance of the gland, to secure drainage of all pockets of pus, etc. Then tampon the incision into the pancreas, using a large rubber tube for drainage in the center of the tampons. In some cases a counter-incision in the left loin will be desirable, as in cases of subacute pancreatitis (page 656). Should a hypogastric incision have been made at first through inadvertence, it should be plugged temporarily with gauze, and may be used at the conclusion of the oper-

ation for pelvic drainage, if this seems indicated; but in many cases it is better to close it entirely. The biliary tract should be drained except for very positive contra-indications,

After-treatment does not differ materially from that employed in other acute lesions of the upper abdomen. If the patient does well, it may be expected that there will be rather free discharge from the drains, and that after four or five days some evidence of pancreatic fluid will be found on the dressings. Should meteorism, vomiting, fever, etc., reappear some days after operation, the patient having done well meantime, the surgeon should suspect either a fresh pancreatic hemorrhage or at least that his drains are blocking rather than aiding the evacuation of sloughs or pus from the pancreas; in such circumstances a new operation is indicated, to establish better drainage, as recommended by Lerchie and Arnaud (1909); but this should not be undertaken until lavage of the stomach and large bowel have proved ineffectual in arresting the symptoms. In some cases, on the other hand, we believe that too early removal of the gauze tampons is responsible for the spread of the pancreatic secretions and the development of a fatal peritonitis. These secretions require much denser adhesions than do ordinary purulent exudates to keep them from breaking through the drainage tract into the healthy peritoneum.

After the first few days the case will resemble one of subacute pancreatitis, so that a more detailed account of the after-treatment may well be postponed until that subject has been considered.

Subacute Pancreatitis

(*Abscess and Gangrene of the Pancreas*)

Abscess or gangrene of the pancreas usually arises as a sequel of acute inflammation of the gland; very seldom is abscess a primary condition, and almost never does gangrene occur except during the subacute stage which succeeds to an attack of "acute hemorrhagic pancreatitis." Some writers attempt to distinguish between *suppurative pancreatitis* and *abscess of the pancreas*. Robson, for instance, compares the former to acute suppurative mastitis and the latter to mammary abscess; but though such a distinction is quite proper theoretically we doubt the ability of surgeons to make a clinical distinction with the means at present at our command; and it is, we believe, also impossible to distinguish between the *suppurative* and *gangrenous* forms of the disease.

The **diagnosis,** in fact, rests more upon the *clinical course* (page 644) of the affection, and upon the *physical signs,* than upon the symptoms which the patient presents. The evidences of deep-seated swelling in the epigastric region, following upon an acute peritoneal attack, always should make the surgeon suspicious of pancreatic trouble; but the physical signs often are very like those of *subphrenic abscess* (page 402), which condition indeed may be due to pancreatic disease. Among the forty-four cases of suppuration in the lesser peritoneal sac studied in 1904 by Michel and Gross twenty-four were certainly, and four probably, caused by pancreatic disease, six by affections of stomach and esophagus, one by perforation of the colon, seven by splenic affections; while in two the cause was undetermined.

Pancreatic abscesses may reach a large size: Fasano (1908) reported one containing four liters of pus, and Coenen's (1910) fifth patient had one which contained more than a liter and a half. These abscesses have a tendency to point (1) in the lumbar regions (Brentano and Rotter have reported cases pointing simultaneously in both loins); (2) anteriorly in the abdominal region; or (3) in the left thoracic region (Guinard). In the first instance the signs of *perinephric abscess* will be simulated, and in the third, those of *pyopneumothorax* or *subphrenic abscess;* while those pointing ateriorly may be mistaken for subphrenic abscess due to subacute perforation of the stomach. In all cases however, there usually will be the history of sudden hyperacute onset, with collapse, peritoneal symptoms, etc., and then as the patient gradually recovers from the first violence of the attack there will be the development of these secondary signs of upper abdominal or subphrenic suppuration; and it is on the clinical history of the disease, *plus* these physical signs, and not on one or the other alone, that a diagnosis must be based.

Treatment consists in evacuation of the abscess and drainage of the pancreas, with removal of such sloughs as are already detached. The abscess should be approached where it is about to point; but if the surgeon wishes to save his patient's life he must not wait for subcutaneous fluctuation to enable him to determine this point. In many cases it will be best to make an exploratory epigastric incision, so soon as it is determined that some form of subacute pancreatitis is present, and then to make a counter-incision in the loin or elsewhere, as may be shown by this exploration to be best.

1. *The Abdominal Route.*—After opening the abdomen through the left rectus, in the epigastric region, the intestines are walled off with gauze packs, and the pancreas is palpated; if at all feasible, the

lumbar route is now employed for drainage. If this does not seem possible, access to the pancreas is gained preferably through the gastro-colic omentum. If a frank abscess is found, it should be evacuated by aspiration, and, when emptied, the abscess wall should be incised. Sloughs which are entirely loose are then extracted, but those which are still attached even in part should not be detached roughly for fear of exciting hemorrhage or spreading infection. The abscess cavity is then tamponed, a rubber drainage tube being placed in the midst of the gauze packs. The gall-bladder should then be inspected, and if acutely inflamed or containing calculi, should be drained, or removed, unless distinct contraindications exist. Such drainage is better done through a stab wound on the right. The gauze used as coffer-dam to protect the general peritoneal cavity during the operation, is then removed and the abdominal incision is closed not too tightly around the gauze and tube drainage. It is not necessary nor is it advisable to attempt to suture the walls of the abscess cavity or the edges of the opening in the gastro-colic omentum to the parietal peritoneum. The abdominal route is preferred by Desjardins in all cases; but Brentano, who has operated on six patients with pancreatic necrosis, with only two deaths, employed the abdominal route in only one case, which terminated fatally.

2. *The Lumbar Route.*—This we believe to be preferable to the abdominal route whenever it can be employed. As mentioned above the applicability of this route sometimes cannot be determined except by exploratory laparotomy. But if bulging, or even marked tenderness without other signs, can be detected in the left costovertebral angle, an incision as for kidney operations should be made here without previously opening the abdomen. Care is necessary not to penetrate the peritoneum; the dissection is carried beneath the lower pole of the kidney, and by burrowing with the finger toward the middle line, little difficulty should be experienced in locating the pancreatic exudate. The abscess is freely opened, and drained with tube and gauze. As already mentioned Brentano and Rotter have each opened (1909) a pancreatic abscess through both loins simultaneously. The lumbar route has been employed several times successfully by the senior author. (See Plate IX, facing p. 640.)

3. *The Thoracic Route.*—This has been particularly commended by Guinard, by whom it has been employed twice with success. In the first case (1898) he operated two months and a half after the onset of acute pancreatitis on a patient who had already had a discharge of pus through the vagina, with sudden subsidence of the pancreatic

tumor; when this refilled operation was undertaken and the abscess was reached by the usual transpleural route employed for subphrenic and hepatic abscess. (See Hepatic Abscess, page 552.) In his second case (1907) operation was done two weeks after the acute onset, for signs resembling left pyopneumothorax, or subphrenic abscess. Körte (1911) used the transpleural route in three cases, one patient recovering; and Nordmann (1913) also reports a successful case.

Prognosis.—If the condition is recognized and operation is done at the appropriate time, the prognosis is not bad. But the surgeon must be constantly on the alert, so as not to let the opportune time for surgical intervention slip past. As soon as the acute process shows signs of localization, but before hectic temperature, chills, sweats, emaciation, etc., show that septic absorption is going on, the pancreas should be drained (see case report, p. 648). Though cases have been reported in which spontaneous discharge of pancreatic abscesses has occurred (through the stomach, rectum, vagina, and in the lumbar and iliac regions), and though in one or two of these cases the patients have recovered without operation, no such termination should be waited for; and though even sloughs of nearly the entire pancreas have been discharged through the rectum (Trafeyer, Chiari), it is not too much to say that were such an event to occur to-day the patient would recover more by good luck than by good management.

That the mortality after operation in the subacute stage, as reported by many surgeons, is lower than that attending early operation, is not an argument in favor of delay in resorting to operation. It indicates merely that the patients who have survived through the earlier stages of the disease have been less seriously ill from the first or have possessed better recuperative powers. Robson (1907) gave the mortality for acute pancreatitis as 61 per cent., and that for the subacute stages as 36 per cent. Villar (1909) gave 78 per cent. as the mortality for hemorrhagic pancreatitis, 38 per cent. for the suppurative and 49 per cent. for the gangrenous form. Mettin (1912) reported a mortality of 71 per cent., for operations done during the hemorrhagic or suppurative stages; and of 66 per cent. for those done during the stage of necrosis.

After-tretament.—This comprises both *local* and *general* treatment. The outer dressings will require frequent changing at first, but the gauze drainage should not be removed until it becomes loose of itself; when this stage is reached, there is no object, so long as free drainage exists, in keeping the wound widely open. Sloughs may be discharged

from time to time, and the surgeon usually finds that closure of the wound is less rapid than he had anticipated, and that his best efforts will have to be directed not at keeping it open but in encouraging it to heal. Protection of the skin surrounding the wound is best secured by covering it with ointment of zinc oxide. Constitutional treatment is important, the patients becoming rapidly emaciated and requiring stimulants and nutritious food. Antidiabetic diet should be used, consisting almost entirely of fats and albumen, as advised by Wohlgemuth (1910), and sodium bicarbonate should be administered during meals to lessen the gastric acidity, which is an excitant of pancreatic secretion; or pankreon may be given to substitute the pancreatic secretion lost through the fistula. Erepton proved effective in closing a pancreatic fistula in a case of rupture of the pancreas reported by Kroiss (1911); about 100 grams were given daily, 20 grams at a dose, by mouth in much sweetened coffee or warm milk; or by rectum in doses of 50 grams. In this patient, no change occurred in the profuse discharge from the fistula for a week after the administration of erepton was begun; then the fistula closed rapidly in three days and remained healed.

Chronic Pancreatitis

Chronic pancreatitis is an inflammation of the pancreas resulting from bacterial infection not severe enough to produce the acute type of inflammation which has already been studied. The channels of infection in cases of pancreatitis have been considered at length at page 614.

It has been usual to classify chronic pancreatitis as catarrhal or interstitial, according as the ducts or the interstitial tissues are especially affected.

Chronic Catarrhal Pancreatitis.—In this form, which is termed also *sialodochitis pancreatica*, the infection is believed to reach the ducts of the pancreas by way of the duodenum and common bile-duct, for the reasons already set forth (page 617). This form of pancreatitis is considered by Mayo Robson and others to be of frequent occurrence. The number of cases available for microscopical study has been small, necessarily, as death seldom occurs at this stage of the disease, and even if it did attention might not be directed particularly to the pancreas. Some cases, however, have been studied; and while the usual lesions present in catarrhal inflammation of glandular organs have been found (cloudy swelling, desquamation, etc.)

there also have been present interstitial changes, which in our belief, as will be stated presently, are of more import than the strictly catarrhal features of the inflammation.

The *symptoms* of chronic catarrhal pancreatitis, according to Mayo Robson, usually are not to be distinguished from those which he describes as due to chronic interstitial pancreatitis; and the *treatment* is the same.

Pancreatic Lymphangeitis.—Reference to this condition at page 620 has indicated our belief that most cases classed together under the general term of "Chronic pancreatitis" are at first really cases of pancreatic lymphangeitis, the infection being propagated from the gall-bladder and bile-ducts or from the pyloric region of the intestine along their efferent lymph-channels, which come into intimate relation with those surrounding and imbedded in the head of the pancreas (Deaver and Pfeiffer, 1912). Not only the grosser lesions of the gall-bladder and juxta-pyloric regions, such as cholecystitis and gastric or duodenal ulcer, should be considered in this connection, but also those more frequent minor infectious processes which pathologically are termed catarrhal and which usually fail of clinical recognition, except as "indigestion" or "dyspepsia." We are accustomed now to recognize extremely mild grades of inflammation of the gall-bladder by the presence of ever so slight thickening, diminution in lustre, increased opacity, or inspissation and tarry character of its contents. If the same attentive scrutiny were directed to the stomach and duodenum, slight thickening, local puckering or distortion and increased opacity in many cases otherwise obscure might reveal the present or past existence of inflammation. As evidence of previous inflammation we have observed dimpling of the wall, at times suggestive of the starting point of an intussusception. Filmy adhesions, so well described by R. T. Morris (1905) as "cob-webs in the attic of the abdomen," should not pass unnoticed; they are clear evidence of a previous inflammatory process. Less noticeable, but significant when observed, is a peculiar streaked, opaque appearance affecting a very limited portion of the gastro-duodenal wall and often fading away in adjacent peritoneal attachments. Of course the grosser cicatrices of healed ulcers are readily seen, but these less conspicous changes should be looked for, since they indicate in many instances the previous existence of interstitial inflammatory processes. Catarrhal inflammation, fissures, and even early ulcers, may exist without external evidence of their presence; and yet they act as portals of bacterial invasion as proved by the enlargement of the regional lymph-nodes.

The lymphatics of the pancreas have been studied thoroughly by Bartels (1904, 1906, 1907) and the following description is taken largely from his work, and from that of Franke (1911), who has studied the relation of the biliary lymphatics to the pancreas. The pancreas, unlike certain other organs, possesses no great hilum through which pass the afferent and efferent blood- and lymph-vessels. These vessels are distributed to the pancreas in a *more or less segmental manner*. Thus the blood-vessels which supply the head of the pancreas are quite distinct in origin from those which supply the rest of the gland; and these latter, derived from the splenic artery, supply each its own more or less isolated segment, though of course intra-pancreatic anastomoses exist. In a like manner the pancreatic lymph-channels, arising in the interior of the gland, come to its surface by many different and quite distinct trunks. These trunks communicate with various groups of lymph-nodes around the pancreas, the most important of which are thus enumerated by Bartels: the classification is made according to the situation of the lymph-nodes and the portion of the pancreas which they drain.

Group I. The Pancreatico-splenic Group.—These lymph-nodes are situated above and behind the tail of the pancreas in the hilum of the spleen. They lie along the splenic vessels between the layers of the gastro-splenic omentum. The lymph-vessels coming out of the tail of the pancreas run to these nodes.

Group II. The Superior Pancreatic Group.—These lymph-nodes lie along the superior border of the pancreas and receive the lymph-vessels coming from the upper portion of the body of the pancreas. There is no sharp differentiation between this and the pancreatico-splenic group and their vessels anastomose freely. Included in the superior pancreatic group are the nodes lying behind the pylorus, often spoken of as the retropyloric nodes. In addition to these connections the lymph-vessels from the upper border of the pancreas have connections with the superior gastric nodes (including the cardiac) and also with the hepatic nodes.

Group III. The Inferior Group.—This consists of a small number of lymph-nodes situated along the lower border of the pancreas. From the lower portion of the body of the pancreas lymphatic vessels run to these nodes and also to the aortic, mesenteric and mesocolic groups.

Group IV. The Pancreatico-duodenal Group, Anterior and Posterior. These lymph-nodes lie around the head of the pancreas and receive vessels from the pancreas and from the duodenum.

In addition to indirect communication by way of the lymph-

atic nodes there is also direct communication by anastomosis between the duodenal and pancreatic lymphatic vessels. In addition to this direct communication there are also anastomoses between the pancreatic lymph-vessels and those from the duodenum running to the mesenteric nodes. Of especial interest from the clinical standpoint is the communication shown to exist between the vessels from the head of the pancreas and a lymph-node situated between the portal vein and the common duct. Lymphatics run from the liver to this node.

It is in this particular that Franke's work supplements that of Bartels. Franke showed by Gerota injections that the lymphatic vessels of the gall-bladder run to nodes which lie to the left of the head of the pancreas near the common duct. On the way the greater part of these vessels are in relation with a node, which, when present, is situated at the neck of the gall-bladder. By injection from the gall-bladder Franke filled a plexus of lymph-vessels situated on the posterior surface of the head of the pancreas.

These four groups of lymph-nodes receive tributaries not only from corresponding portions of the pancreas but also from neighboring viscera, the spleen, stomach, left adrenal, liver and duodenum. The efferent vessels from the pancreatic nodes run to the parietal lymph-node groups of the abdominal cavity.

Lymphatic-born infection from these areas in order to reach the pancreas must in most cases stem the efferent current from the pancreas and force the valves. Here, as elsewhere in the body, the lymphatics are wonderfully efficient in preventing this outcome. Only when the intercommunications of the pancreatic lymphatics with those of adjacent organs are most intimate, short in their course, and unprotected by interveining lymph-nodes, does peril arise. Such an extremely intimate relationship Bartels has shown to exist between the lymphatics of the head of the pancreas and the adjacent duodenum; and more recently Franke has demonstrated that the same is true of the lymphatics coming from the gall-bladder. Should thrombo-lymphangeitis occur in any of these vessels as the result of infection originating in the duodenum or gall-bladder, reversal of the lymph-current may occur in the pancreatic lymph-channels, in an effort to establish a collateral circulation; and in this way infection may be propagated from the gall-bladder, bile-ducts, or duodenum to the pancreas. A sufficiently severe infection might not even wait for reversal of the lymphatic current, but might rapidly invade the pancreas and infect not only the lymphatic tissues but the surrounding structures as well. The intrinsic lymph-vessels of the pancreas run in the inter-

lobular septa, and it is here that the effects of this type of inflammation should be most manifest; and in accordance with this explanation is the fact that it is the interlobular form of pancreatitis which is associated with inflammatory lesions of neighboring viscera, in contrast with the interacinar sclerosis, which, as pointed out by Opie, appears to bear no such relation to local inflammation. It is true of course that the interlobular distribution of the inflammatory changes might be attributed to infection brought by the blood-vessels or extending along the pancreatic ducts, since all these structures run in the interlobular tissues. But if the infection were propagated by these channels it should be diffusely distributed throughout the gland; and this is not the case in the earlier forms of the disease. It has been observed by all surgeons that the earlier forms of pancreatitis found at operation in connection with gall-bladder or duct disease involve only the head of the pancreas or perhaps only a portion of the head; and the "triangle" of duct-born infection described by Desjardins (see page 618) loses much of its interest when we consider that a *segmental pancreatic lymphangeitis* explains the limitation of the infection to the head of the pancreas in a much more rational manner. Thus *the distribution of the pancreatic inflammation corresponds to the lymphatic distribution*, which, as pointed out above, is irregularly *segmental*. It does not correspond to the duct distribution, which ramifies by dichotomous division from the main accessory ducts. If the infection were ductal in origin, the gland should be symmetrically involved. This point was emphasized by Arnsperger (1911).

The swellings of the head of the pancreas which are so frequently encountered by the surgeon must be different from the varieties of chronic pancreatitis described by the pathologist. Kehr (1909), on clinical grounds, surmised that such a difference exists. "Chronic pancreatitis," which is characterized by interlobular or interacinar deposits of fibrous tissue, can be no more curable than chronic nephritis or cirrhosis of the liver. But it is characteristic of the pancreatic swellings associated with biliary disease that they subside with the disappearance of the biliary infection; such swellings therefore must be due to edema, congestion, and absorbable infiltrates. The subsidence of such a swelling in the head of the pancreas after cure of the primary infection is analogous to that which occurs in the treatment of the primary foci of lymphatic infection elsewhere in the body. In many cases of what we term pancreatic lymphangeitis it is possible to demonstrate the chain of infection: infected gall-bladder, enlargement of the cystic node, enlargement of the nodes around the head of the pan-

creas, and swelling of the head of the pancreas, which corresponds to the regional lymphatic distribution. Since we have been looking for these lymph-nodes, it is remarkable how constantly they have been found in this condition. One node which appears to be especially constant in position and enlargement is situated just to the right of the choledochus where it passes beneath the duodenum.

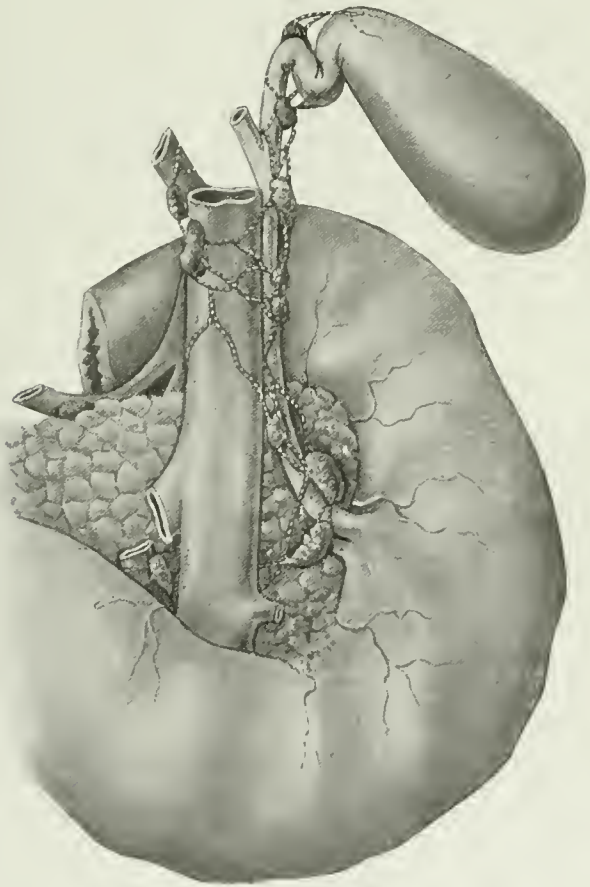


FIG. 161.—Lymph-nodes of the Bile-ducts and Head of the Pancreas. (*Modified from Cunéo.*)

We have spoken of this condition of pancreatic lymphangeitis hitherto chiefly in connection with gall-bladder lesions, because in such cases it is most easily recognized; but it is our belief that lymphatic infection from the pylorus and duodenum may play an important part in the pathogenesis of chronic pancreatitis. Not a few instances of the association of duodenal ulcer and pancreatitis are on record, while

catarrhal duodenitis may also be a factor. It is possible that still other organs may at times furnish infection to the pancreas; but at present the biliary tract and the duodenum seem to be the chief sources of infection, on anatomical, clinical and pathological grounds.

A brief outline of the following case suggests that disease of the appendix may exert an influence on the pancreas.

Operation was performed for gangrenous appendicitis in a female child who had suffered with chills and high fever, the chills occurring two or three times in twenty-four hours. The appendix was removed. Chills and fever persisted and the child developed acute abdominal pain. A second operation was performed and a small abscess was found completely encapsulated in the mesentery of the ileum. Pancreatic lymphangeitis was present, the pancreas itself being greatly edematous and infiltrated. Death occurred ten days after the second operation. Except for the pancreatic lymphangeitis above referred to, no lesion was found in the abdomen at autopsy.

The following are brief notes of a case in which Dr. E. G. Alexander operated, in the service of Dr. H. C. Deaver, Episcopal Hospital:

Adult male, aged 23, suffering with acute appendicitis, chills and high fever. The appendix was removed; no pus and few adhesions were found. Seventeen days after operation the patient had a marked chill, was reoperated on and a large mesenteric abscess drained. Death occurred five months after the second operation. Pathologic diagnosis was multiple abscess of liver, chronic fibroid peritonitis, post-operative peritonitis in region of appendix, acute splenic congestion, acute parenchymatous nephritis, suppurative cholecystitis and abscess of the head of pancreas.

In these cases it appeared that the source of the peripancreatic and pancreatic inflammation was the infected appendix and that the path of metastasis was retroperitoneal and through the lymphatics along the cystic and common ducts.

It must be conceded that in many inflammatory conditions of the abdomen there is a retroperitoneal lymphangeitis which is fraught with the possibilities of injury to the pancreas since this lies almost directly in its path.

That this condition of pancreatic lymphangeitis has not been recognized often at autopsy is no doubt to be explained in this way: the lesions are comparatively slight, and, even if the pancreas is examined microscopically, they can be confused easily with that autodigestion of the organ which ensues at once after death and so often renders examination of pancreatic tissue unsatisfactory; moreover the clinician has not called attention to the desirability of a search for minor pathological

alterations. The disease is not itself immediately fatal; if it is relieved by the operative treatment instituted the pancreas may return to its normal state; while if unrelieved it will progress to true chronic interstitial (interlobular) pancreatitis, with deposition of fibrous tissue.

At the present time the *diagnosis* can be made only by the surgeon who palpates and inspects the gland during operation. The *symptoms* are those of the primary disease—cholecystitis, cholelithiasis, etc.; and the *treatment* involves only proper treatment of the causative lesion.

The following case is cited to show the extent to which the disease may progress. The pathological changes are too far advanced and the etiology too obscure to prove its lymphatic origin in this instance. The glandular involvement, however, is similar in many respects to that observed by us in a considerable number of earlier cases where the considerations just mentioned lead to the belief in a lymphatic origin.

D. G., aged thirty-seven years. Admitted to the German Hospital, January 6, 1912. Died January 19, 1912.

Chief complaint, cramps in upper abdomen, radiating into flanks and to right side of back.

Family History.—Mother died of tuberculosis; otherwise negative.

Personal History.—Tea, coffee, and tobacco in excess; alcohol in moderation; denies venereal diseases. In March, 1910, he was admitted to the Pennsylvania Hospital suffering with pain beneath the right costal margin radiating to the back. The pain was worse two or three hours after eating. Constipation was a marked symptom, and he had lost considerable weight. At operation cholecystitis was found and cholecystostomy performed. In July of the same year he was again operated upon for recurrence of symptoms and cholecysto-duodenostomy performed. In June, 1911, he was again in the hospital with the same symptoms plus jaundice, chills and fever. Cholelithiasis was diagnosticated, but he was not operated upon. In November, 1911, after intermittent attacks resembling those of stone in the common duct he came to the German Hospital and was again operated upon. Chronic pancreatitis, chronic cholecystitis, and a stone in the common duct were found. The stone was forced into the duodenum. The cholecysto-duodenostomy opening had become obliterated; cholecysto-duodenostomy performed. Recovery was uneventful, and he was improved for a few weeks, when his trouble recurred. More recently he has been having chills, fever, and jaundice.

Physical Examination.—A poorly nourished man of nervous appearance. Skin moderately jaundiced. Head and chest negative.

Abdomen: Scar of old incision through upper right rectus. In this region and in the epigastrium there is marked tenderness on pressure, but no rigidity or mass.

Urine shows a very faint trace of albumin, and a few hyaline casts, otherwise negative.

Blood: Hemoglobin, 73 per cent.; erythrocytes, 4,050,000; leukocytes, 7,300 (polymorphonuclear neutrophiles, 73.5 per cent.; lymphocytes, 26 per cent.; eosinophiles, 0.5 per cent.); coagulation time, 8 minutes.

Stool: Dark green and practically non-odorous; fluid; neutral reaction; a trace of bile, occult blood positive to benzidine and guaiac tests. Azotorrhea and steatorrhea marked.

Cammidge reaction positive.

Operation, January 13, 1912.—Dr. Deaver. Ether anesthesia. Excision of old scar in upper right rectus region; adhesions of stomach and right lobe of liver to incision; hepatic flexure of colon adherent to right lobe of liver; great omentum adherent to lesser omentum:; small whitish tubercles found on small intestines; some adhesions between ascending colon and parietal peritoneum; liver covered with a few whitish nodes which are subserous; old cholecysto-duodenostomy still patulous; head of pancreas enlarged and hard; felt semicystic; foramen of Winslow, though occluded, was forced open; small glandular enlargement at junction of the supra- and retroduodenal portions of the common duct; head of pancreas opened by inserting scissors and withdrawing them opened (Hilton's method). Much hemorrhage followed; this was controlled by a piece of selvage gauze and one suture of

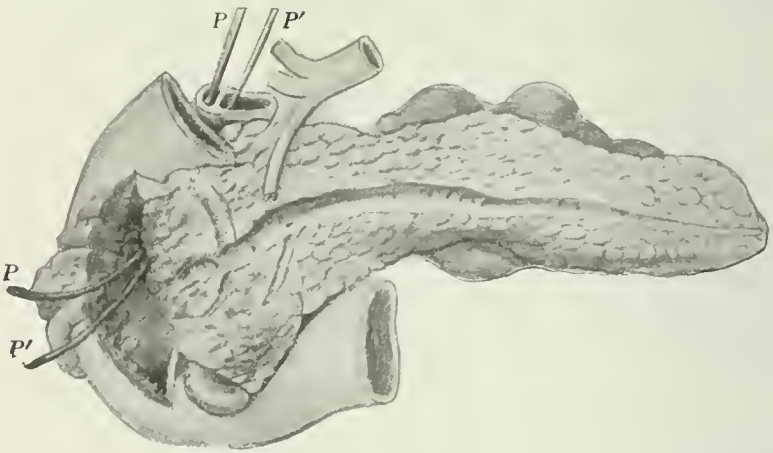


FIG. 162.—Anterior View of Pancreas, with Duct of Wirsung Opened Throughout its Length and a Window in the Duodenum. *P P*, Probe from Cystic Duct through Gall-bladder and Cholecysto-duodenal Anastomosis. *P'P'*, Probe from Dilated Common Duct into Duodenum through Ampulla of Vater. (Case of D. G., page 665.)

iodin gut; duodenum incised and old cholecysto-duodenostomy proved patent; much bile-stained material escaped from opening; head of pancreas seemed to be obstructing the duodenum posteriorly from pressure; duodenum closed with catgut baseball suture and a Lembert of linen thread; pancreas at site of incision sutured to parietal peritoneum; one cigarette drain anterior to gastrohepatic omentum; one rubber tube at foramen of Winslow; another rubber tube anterior to this and wound closed in layers.

Following the operation there was profuse brownish drainage from the wound. The jaundice disappeared. He became more emaciated and gradually weaker and died January 10, 1912.

Autopsy through the incision permitted removal of the pancreas and adjacent tissues. The head of the organ was somewhat smaller than normal, and appeared

necrotic. The selvage gauze in the pancreatic wound had ulcerated into the duodenum at the site of the ampulla of Vater. A fistula existed at the site of the exploratory incision into the duodenum. A striking feature was the occurrence of many enlarged lymph-nodes along the upper margin of the pancreas. A few were present along the lower border, and in relation to the head both anteriorly and posteriorly where embraced by the duodenum.

Microscopically the tail of the pancreas was the seat of a moderate grade of interlobular sclerosis. The parenchyma was mostly degenerated, probably due chiefly to autodigestion. The islands of Langerhans were intact. The head of the organ showed marked interlobular pancreatitis and an acute suppurative process superimposed. The parenchyma was necrotic. The islands of Langerhans persisted but were involved in the acute necrotic process. A section made through a small villous patch at the junction of the gall-bladder and duodenum (at the cholecysto-duodenostomy opening) showed a marked glandular proliferation which had begun to invade the deeper tissues. The process had not extended more than 0.5 cm. from its starting-point, and no metastasis could be demonstrated. The lymph nodes showed chronic and acute hyperplastic lymphadenitis.

Chronic Interstitial Pancreatitis.—Attention was first prominently drawn to this conclusion by Mayo Robson in 1900. It is infectious in origin; and as we have pointed out in the preceding pages, the gall-bladder and bile-ducts are the chief primary foci.

Of seventy-nine patients in Lankenau Hospital with chronic pancreatitis, seventy-two (91 per cent.) showed evidence of infection in the bile-passages; forty-two (53 per cent.) had gall-stones, and in thirty (38 per cent.) there was non-calculous inflammation.

W. J. Mayo (1809) reported 359 cases of pancreatitis, 86 per cent. of which were accompanied by gall-stone disease. Pancreatitis in some form was present in 7 per cent. of all patients where gall-stones were found in the gall-bladder; and in 27 per cent. of cases of stone in the common duct marked pancreatitis was present. This is in favor of the view held by Maugeret, and referred to at page 621, that the occurrence of pancreatitis results in stenosis of the bile-ducts, with resulting arrest of calculi in the common and hepatic ducts; but it also indicates that bile-duct infection predisposes to pancreatitis. A vicious circle is formed in this way.

Pathogenesis.—The *avenues of infection* in pancreatitis have been sufficiently discussed (page 614). From all that is known we believe it is highly probable that in the vast majority of cases of chronic pancreatitis the infection occurs through lymph-channels and that the earliest stage of the disease is a pancreatic lymphangeitis (p. 659).

Predisposing Causes. *Age.*—The age at which pancreatitis is said to occur depends upon whether the reported cases are from

operative or autopsy records. This is readily demonstrated by comparing Opie's cases with ours.

	Opie	Deaver
Under 30 years	3 cases	4 cases (non-calculous)
30 to 40 years	2 cases	11 cases
40 to 50 years	9 cases	11 cases
50 to 60 years	11 cases	8 cases
Over 60 years	5 cases	0 cases
Total	30 cases	34 cases

More than two-thirds of Opie's cases (autopsy records) occurred between the ages of forty and sixty. In our series, all but one being operative cases, two-thirds occurred between the ages of thirty and fifty.

Sex.—In thirty-seven of our patients with chronic pancreatitis without gall-stones, twenty-two were males and fifteen females. Opie reported seventeen males and thirteen females among thirty patients. Taken in conjunction with the well-known preponderance of gall-stone disease in women, these figures indicate that in men there frequently must be some other source of infection, probably the pylorus and duodenum.

Pathology.—Opie (1908) divided cases of chronic interstitial pancreatitis into two classes, *interlobular* and *interacinar*, with another form of degeneration, *lipomatosis*, common to both classes, and in each case the latest stage of the disease. While both kinds of pancreatitis consist essentially in the deposition of newly formed connective tissue, the difference between them is due to the position in which the fibrous tissue is laid down, and this pathological difference corresponds to difference in etiology and symptomatology.

From a surgical standpoint chronic interlobular pancreatitis is the only form of interest, as interacinar pancreatitis is not amenable to operative treatment.

Chronic Interlobular Pancreatitis.

The interlobular connective tissue is not well marked in the normal pancreas, consequently the lobules are not sharply defined. In interlobular pancreatitis, however, these bands of connective tissue become greatly increased in size and density, accentuating the lobulation. A "typical" case of chronic interlobular pancreatitis has the following characteristics: In most cases, at least early in the disease, the head of the pancreas is the part affected. The probable reasons for this we have already stated in the discussion on pancreatic lym-

phangeitis. Only very late is the entire pancreas involved. The portion of the pancreas affected is enlarged and hard with a nodular surface; on section, tense bands of fibrous tissue traverse the cut surfaces in relatively the same manner as the normal interlobular framework, but the increase in amount and density results in the formation of well-marked lobules. From this classical type, readily recognizable at operation or autopsy, all grades of interlobular change occur down to those cases in which the pancreas shows no macroscopic evidence of disease.

Microscopical Examination.—The most striking feature of this form of pancreatitis is the escape of the islands of Langerhans from any involvement in the sclerotic process. It is only very late in the disease, if at all, that they are involved. The disposition of connective tissue is most marked in the interlobular tissue, although, as the disease advances, there is a certain amount of invasion of the parenchyma with the replacement of normal parenchymatous cells by fibrous tissue. Later, this replacement may become so well marked that nothing of the parenchyma is left but the islands of Langerhans, which appear proportionately in greater numbers on account of the shrinkage of the connective tissue. Even at this late stage the islands of Langerhans are intact and functionating, as is shown by the non-interference with carbohydrate metabolism. When glycosuria does occur, as it may occasionally, late in the disease, it is due to interference with the blood supply of the islands of Langerhans from the pressure of the contracting fibrous tissue on the arteries.

Lymphoid cells, plasma cells and eosinophiles are numerous in the newly formed connective tissue, an evidence of active inflammatory change.

If this form of pancreatitis was caused primarily by obstruction or infection of the ducts, the parenchyma surely would be involved very early in the disease, since this is the active secreting portion of the pancreas connected with the excretory ducts. As already stated, however, parenchymatous changes occur late in the disease, the interlobular connective tissue being affected long before the parenchyma, and the islands of Langerhans remaining unaffected.

A certain amount of obstruction of the pancreatic ducts may result in this form of pancreatitis, from pressure by the interlobular fibrotic changes. In this way a condition is produced somewhat similar to that seen in experimental obstruction of the pancreatic ducts, which in animals results in a form of interlobular pancreatitis without involvement of the islands of Langerhans.

Chronic Interacinar Pancreatitis.

The deposition of newly formed connective tissue may take place within instead of between the lobules of the gland, with the result that the parenchyma is largely replaced by fibrous tissue, which is *more or less evenly distributed throughout the substance of the pancreas* without accentuating the lobulation. This form of pancreatitis frequently is associated with sclerotic changes in the arteries; and as a consequence the nutrition of the islands of Langerhans is affected early, and diabetes often results. The process sometimes seems to start in the islands of Langerhans. Sixty-seven of ninety cases of diabetes showed interacinar pancreatitis, and in sixty-five the arteries of the pancreas were sclerotic (Cecil, 1911). As a consequence there is interference with the carbohydrate metabolism and the appearance of glycosuria (pancreatic diabetes). This condition contraindicated operation; therefore interacinar pancreatitis is of secondary importance in a work on surgery.

Lipomatosis.—This condition consists in the deposition of large amounts of fat in the connective tissue. It may occur in either form of pancreatitis. Its cause and significance are not understood and while it does occur in fat people, obesity is not a necessary factor in its production, as it also occurs in patients who have lost a good deal of weight.

Symptoms and Diagnosis.—The extremes of opinion in respect to the diagnosis of chronic interstitial pancreatitis are represented by the pessimism of Opie and the optimism of Robson and Cammidge. The former says that "chronic pancreatitis is rarely accompanied by such definite symptoms that its recognition is possible during life," while the latter hold that "from the information obtained from a careful examination of the patient, a knowledge of the history of the case, and the results of a chemical and microscopical examination of the excreta, a correct opinion may be formed in a large majority of instances." Our feeling in this matter is midway between the two extremes. The diagnosis has been made often enough to demonstrate that it is not too difficult to attempt; but it is equally true that our present criteria are too uncertain and inconstant to warrant a claim for great accuracy. There are no pathognomonic symptoms. Chronic pancreatitis is so often associated with disease of surrounding organs that it is difficult to separate the symptoms due to the accompanying disease and those due to the pancreatitis. Of course disease of the bile-ducts, particularly the common duct, suggests the probable presence of pancreatic disease, although the symptoms referable to each condition cannot be differentiated.

The factors to be considered in making a diagnosis are:

1. The clinical history.
2. The physical examination.
3. Special tests designed to show disturbance of pancreatic function.

The following symptoms are those obtained in the analysis of a series of cases of chronic pancreatitis (under the senior author's care) in which there were no gall-stones present at the time of operation. As about one-third of these patients had demonstrable changes in the gall-bladder, there may be a certain admixture of symptoms referable to involvement of the bile-passages.

Another point to be remembered is that these cases represent patients that underwent operation, consequently one would expect symptoms to be more marked than in a series of cases studied post-mortem. This point may serve as a possible explanation of the extremely divergent views on the possibility of diagnosis, the pathologists' view being advanced by Opie and the surgeon's by Robson.

Previous History.—Particular attention should be paid to a history of previous gastro-intestinal trouble or habits of eating and drinking likely to cause it; also to the occurrence of any of the infectious diseases habitually followed by cholelithiasis. If disease of the bile-ducts and gall-bladder has preceded the pancreatic inflammation, the early history presents the symptoms of that disorder, with perhaps frank attacks of biliary colic.

The relation of age and sex to chronic pancreatitis has been discussed (page 667).

Symptoms.—The onset of symptoms was sudden in two-thirds of the cases and gradual in one-third; but these figures refer especially to exacerbations, premonitory symptoms being present in the majority of instances.

The symptoms usually associated with chronic pancreatitis are pain, nausea and vomiting, icterus, fever and loss of weight. These are common to various upper abdominal diseases and do not of themselves, even when all are present, constitute a characteristic symptom-complex.

Pain is a leading and most constant symptom. It was present in over 90 per cent. of our cases. This, of course, represents operative practice. The general practitioner must see many patients in whom pain is absent or so slight as to be negligible.

The *character* of the pain is not constant. It varies from a dull

ache with a sense of fullness or oppression in the epigastrium to sharp lancinating pain like gall-stone colic. In our series it was severe in twelve cases, moderate in twenty-one, absent in three and not mentioned in two histories. Nearly one-third of the patients had *distinct attacks of colic* and in the majority of these the gall-bladder was diseased. In one instance several stones were passed before operation although no stones were found when the abdomen was opened. It is probable therefore that colicky pain seldom occurs in chronic pancreatitis unless the bile-passages are involved. Colic from the passage of pancreatic calculi is indistinguishable in character from biliary colic, but the pain of chronic pancreatitis is the result of inflammatory changes and is not a colic. Yet Archibald (1910), having observed on several occasions attacks of colic in patients in whom at operation no biliary lesions were demonstrable, suggests that these colicky pains may have been caused by retrojection of bile into the pancreatic duct, his idea being that during the long periods of fasting which patients with chronic pancreatitis undergo, the back pressure of bile is so augmented as to cause it to enter the pancreatic duct, though it is insufficient to force the sphincter of Oddi, except when this may become relaxed by physiological stimulation when chyme enters the duodenum (page 38). Archibald adds that the condition of fasting referred to above, as being likely to leave the sphincter closed, is especially frequent in chronic alcoholics, among whom this form of pancreatitis is so frequent.

According to Sailer (1910) the administration of a large amount of glucose to test the assimilation limit is particularly distressing to patients with chronic pancreatitis.

Michel (1911) quotes Desjardins, Vautrin and Dieulafoy as stating that insidiousness is the dominant characteristic of the pain of chronic pancreatitis.

The site of pain varies as does the character. It was in the epigastrium in 45 per cent. of the cases in our series, beneath the right costal margin in nearly 40 per cent., beneath the left costal margin in 3 per cent. and in the lumbar region in 6 per cent.

Radiation of pain from the original site occurs in nearly 90 per cent. of these cases, to the epigastrium, the back, the shoulders, etc. Mayo Robson says that the attacks of pain have been mistaken for those of angina pectoris.

No definite relation to eating, drinking, or any particular food could be determined, a point of possible value in the differential diagnosis from gall-stone disease, gastric or duodenal ulcer.

From these figures it is evident that the pain occurring in the

course of chronic pancreatitis is not constant in character, position or radiation, nor distinctive enough in any way to differentiate it from the pain of other abdominal diseases.

Nausea and Vomiting.—More than half of our patients gave a history of vomiting at some period of the disease and about one in four was nauseated but did not vomit. In four of Opie's cases, vomiting was so persistent and prolonged that the gastro-duodenitis causing it was considered the starting-point of an ascending infection of the pancreatic duct resulting in chronic interlobular pancreatitis. In none of our cases was the vomiting persistent, but the frequency with which it occurred indicates its importance as a symptom in the advanced type of the disease. The character of the vomitus is not distinctive; it consists of stomach contents, bile and mucus.

Jaundice.—Twenty-eight per cent. of the patients were jaundiced on admission, 34 per cent. gave a history of previous attacks, and 38 per cent. never had been jaundiced.

Jaundice is not a symptom of great value in differential diagnosis because of the various factors capable of causing it. Gall-stones or inflammation of the bile-passages so frequently accompany pancreatitis that this explanation of the occurrence of jaundice is acceptable in a large proportion of cases and Opie considers it adequate in practically every case. However, the problem is not so simple. A certain number of cases of pancreatitis with jaundice occur without any demonstrable lesions in the biliary system. The relation of the common bile-duct in its lower third to the head of the pancreas offers a reasonable explanation under these circumstances. In about two-thirds of instances the lower end of the common duct passes through the substance of the head of the pancreas. The occurrence of jaundice in these cases is explained by the mechanical effect of swelling of the gland causing obstruction to the flow of bile. This factor also explains the persistence of jaundice after the passage of a stone from the common duct. The stone causes obstruction and inflammatory changes resulting in jaundice and pancreatic lymphangeitis. The latter in its turn causes swelling of the head of the pancreas, which by pressure on the common duct keeps up the obstruction after the stone has been passed.

The fact that in about one-third of instances the common duct passes behind and not through the head of the pancreas explains why in a definite proportion of cases of well-marked pancreatitis jaundice does not occur.

The degree of jaundice varies from a slight tinge to the "black jaundice" of the older writers, which was supposed to be diagnostic

of malignant disease. Various observers have attempted to differentiate jaundice caused by pancreatitis from that due to other conditions, but without conspicuous success. Yet it seems to be a fact that jaundice from pancreatic obstruction is less subject to variation than that due to stone in the common duct. Michel (1909) said the characteristics of jaundice in pancreatitis are variable intensity at the onset, then progressive deepening without special pain. Kehr (1909) said that obstruction from a calculus lodged in the upper part of the common duct is accompanied by variations in intensity and by intermittence of jaundice because of the length and mobility of the common duct; but obstruction in the lower part of the common duct, particularly the ampulla, causes continuous jaundice without great remissions. This latter variety, therefore, is very difficult to distinguish from jaundice due to pancreatic disease.

These observations simply prove that jaundice is a symptom upon which no great stress can be laid in making a differential diagnosis of upper abdominal lesions.

When jaundice is continuous and associated with rapid wasting and loss of strength, the clinical picture is that of carcinoma of the head of the pancreas. In 13 per cent. of our cases jaundice was continuous, in the others, intermittent. Intermittent jaundice, pain and fever may occur in the course of chronic pancreatitis and simulate stone in the common duct with Charcot's hepatic intermittent fever.

The *relation of pain to jaundice* in our cases was indefinite. Four times the onset of jaundice was not accompanied by pain. Pain preceding the onset of jaundice may be due to gall-stone colic, but as a rule the pain is less severe and not of a colicky character.

Loss of Weight and Strength.—Emaciation and loss of strength are fairly constant accompaniments of pancreatitis and always manifest themselves when the degree of sclerosis is sufficient to interfere with pancreatic function. The obstruction to the excreting ducts diminishes the amount of secretion reaching the intestine, resulting in imperfect digestion and mal-assimilation. Another factor is restriction of diet, voluntarily or by the advice of a physician, in the attempt to control the symptoms of indigestion. At times impairment of appetite is responsible for decreased intake of food, though loss of appetite is by no means a constant accompaniment of pancreatitis. It was mentioned in about one-fifth of the cases in our series. If jaundice is present biliary intoxication may be a factor in the production of emaciation.

Loss of weight was noted in more than half of our cases. The state of nutrition at the time of operation was poor in 34 per cent., good

in 25 per cent. while 16 per cent. of patients were obese; in 24 per cent. the state of nutrition was not mentioned. Loss of strength follows loss of weight.

Very rapid loss of weight may take place in some instances, particularly when jaundice is a marked feature. Cachexia may be more rapid and extreme than in malignant disease. Moynihan (1909) reported a case where a patient lost 26 pounds in three months; Mayo Robson (1907) reports patients losing 42, 55 and 110 pounds; Chauffard (1911), a patient losing 77 pounds in six months; and Terrier (1906), a patient losing 16 pounds in one month and 44 pounds in eight months.

These are, however, exceptions, and the usual case does not exhibit such marked wasting until a late stage of the disease.

Fever.—Fever is not such a prominent symptom in the prolonged course of chronic pancreatitis as most figures would seem to indicate. As a rule the temperature is normal. Hyperpyrexia is present only during exacerbations. Of our series 13 per cent. gave a history of chills and sweating. At the time of operation 34 per cent. had a temperature between 99° and 100° F. In 10 per cent. it was between 102° and 103° F.

Bowels.—Constipation is the rule; half of our patients suffered with chronic constipation and in more than one-third constipation was a feature of the attacks. In only 13 per cent. was there a history of diarrhea.

From this it must be seen that caution must be used in employing as an aid to diagnosis the classical description of "frequent bulky motions, pale in color, offensive, and obviously greasy." As stated by Robson and Cammidge, such stools are present only in advanced conditions. The stools are likely to be clay colored even when bile is present. If bile is persistently absent to laboratory tests of the duodenal secretions the condition is more apt to be carcinoma of the pancreas than an inflammatory affection.

Diabetes.—Glycosuria indicates involvement of the islands of Langerhans in the sclerotic process. It is a rare symptom in interlobular pancreatitis and is then a sign that the destruction of the parenchyma has advanced to such an extent that the fibrous tissue has either encroached directly on the islands of Langerhans or has, by contraction, seriously interfered with their blood supply. Only 5 per cent. of the cases of our series showed glycosuria. Sugar may appear in the urine during an exacerbation and clear up on subsidence of the inflammation This is a threat of oncoming diabetes and should not be overlooked. Of equal significance is the presence of

alimentary glycosuria, tested by the administration of sugar. Drainage of the infected biliary and pancreatic ducts may cause disappearance of glycosuria when it is associated with interlobular pancreatitis and is of recent development.

In interacinar pancreatitis the symptoms of true diabetes mellitus appear early in the course of the diseae. Recognition of one of the various diseases usually associated with pancreatic diabetes, such as arterial sclerosis, taken in conjunction with the early appearance of sugar in the urine, usually enables a distinction to be made between this form of pancreatitis and that occurring in the interlobular type.

Physical Examination.—The physical examination rarely affords much positive information. It is of more value in excluding other conditions.

Tumor.—During exacerbations there may be epigastric tenderness and rigidity which completely mask the underlying condition. In patients with thin abdominal walls the swollen head of the pancreas sometimes may be palpated between exacerbations of the disease. As a rule, palpation even of a considerably enlarged pancreas is impossible, as in most instances the pancreas is well covered by the adjacent organs.

Korte (1911) examined thirty cadavers with reference to this point. In twenty the pancreas was completely covered, while in ten there was some part covered with omentum only. In six there was ptosis of the colon with exposure of a portion of the head of the pancreas between the liver and colon; in two it was exposed in the median cleft of the liver; in the other two there was marked gastroptosis and the pancreas could be palpated directly beneath the gastrohepatic omentum. In palpating through the abdominal wall these slight exposures of pancreatic tissue rarely are sufficient to give definite results.

When a mass is demonstrable it may be referable to an enlarged gall-bladder or a cancer of the head of the pancreas as well as to chronic interstitial pancreatitis. Even when the abdomen is open the differentiation between carcinoma and chronic pancreatitis may be exceedingly difficult.

Enlargement of the gall-bladder has been observed in a number of cases, but in our cases it was not detected prior to the operation. The liver was noted as enlarged in about 25 per cent. of the cases.

Tenderness.—As most of our patients were operated on during or just after some exacerbation of symptoms there was a degree of

tenderness present in most of them. In eight no tenderness was elicited. In the remainder tenderness was found below the right costal margin in twenty; beneath the left in three; in the mid-epigastrium in eleven; over Mayo Robson's point[1] in three; and in one severe case it was general. Rigidity was observed in the right hypochondrium in nine cases and over the epigastrium in three. There was moderate distention in six cases.

There is then no constant point or area to which tenderness is limited in cases of chronic pancreatitis, although various authors have described them. Desjardins (1898) described a point 5–6 cm. above the umbilicus on a line drawn between it and the gall-bladder area, as the point on the abdomen representing the position where the duct of Wirsung enters the duodenum. Chauffard and Rivet (1911), instead of describing a fixed point, name the area corresponding to the head of the pancreas the pancreatico-hepatic area. This area occupies the lower part of an angle of 45 degrees formed by the midline and a line drawn up and to the right from the umbilicus (Fig. 163).

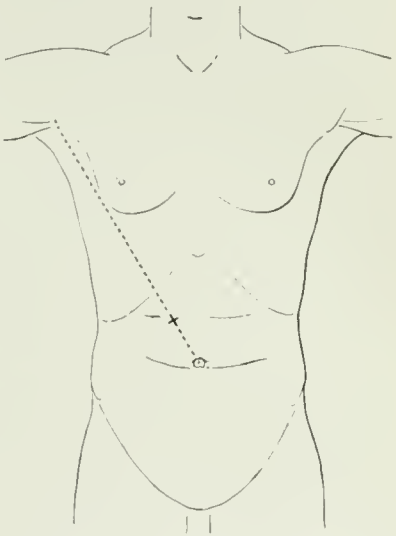


FIG. 163.—The "Pancreatic Point" of Desjardins and the "Pancreatico-hepatic Area" of Chauffard and Rivet.

We think no dependence can be placed on these or similar points and areas of tenderness in differentiating conditions of the upper abdomen.

Blood Examination.—The blood frequently shows secondary anemia which seldom is grave, but may become so if surgical intervention is delayed too long. In about one-half of our cases the hemoglobin was below 80 per cent., and the erythrocytes below 4,000,000. A moderate leukocytosis was present during a few of the more acute exacerbations. Generally the numerical ratio of the leukocytes was unaltered.

Special Tests to Show Disturbances of Pancreatic Function.—The number and variety of laboratory aids to diagnosis show that none of them is infallible. The most important have been discussed at page 626.

[1] This is a point "just above and to the right of the umbilicus" (1907).

Examination of Feces.—The results of examinations of the feces in our cases have convinced us that functional tests of pancreatic activity have not yet reached a stage which warrants much dependence upon them.

The *reaction of the feces* in pancreatic disease may be markedly acid; normally there is an amphoteric reaction.

Examination of the Duodenal Contents.—This is a much surer method for determining the functional activity of the pancreas, than is analysis of the feces. If bile and pancreatic ferments are present, it is not likely that the pancreas is seriously diseased. If, however, the accessory pancreatic duct is patulous, pancreatic secretion may reach the duodenum even in cases of jaundice from obstruction at the papilla of Vater, with pancreatitis.

Examination of the Urine.—It is advisable to collect a twenty-four-hour specimen. Cammidge (1911) recommended a simple mixed diet for two or three days beforehand, rather than any special test diet, as the idea is to measure the capacity for digesting normal food. Routine examination includes tests for the following: Albumen, sugar, urobilin, acetone, diacetic acid, the amount of ammonia nitrogen, the results of the pancreatic reaction, and the presence of calcium oxalate crystals microscopically (Cammidge).

The "pancreatic" or Cammidge Reaction (1901).—As is well known this reaction consisted in the demonstration in the urine, when treated by a rather complex chemical procedure, of certain crystals of a definite morphology and certain solubility characteristics, but of unknown composition, although they were thought to be a derivative of pentose, probably an osozone.

For some years a great deal was written about the Cammidge reaction. Opinions differed as to its value. Some considered it valueless as an aid to diagnosis, others considered it more or less useful and still others apparently depended on the results of the "pancreatic" reaction more than on any other factor. At the present day very few if any surgeons attach any importance to it whatever.

Glycosuria.—This is a sign that appears late in interlobular pancreatitis and is of grave significance. Sugar may appear in the urine during exacerbations and disappear in the intervals. Alimentary glycosuria is another important indication of pancreatic insufficiency that should be looked for.

Calcium Oxalate.—The presence of the characteristic crystals of calcium oxalate is said to be a confirmatory sign of pancreatic insufficiency.

Diagnosis.—Undue prominence seems to be given in the symptomatology of chronic pancreatitis to the description and discussion of the various laboratory methods for determining interference with the function of the pancreas. This may be taken as an indication of the views on diagnosis. The confidence of clinicians in their ability to diagnosis this condition is directly expressed by their faith in the accuracy and reliability of the various tests for pancreatic function.

From the symptoms and physical signs, localization of the disease to the pancreatico-hepatic region almost always can be made, but in the differentiation of chronic pancreatitis from disease of the bile-ducts, or in the diagnosis of pancreatitis accompanying or resulting from gall-stone disease there always is a large amount of doubt, as to the possibility of accurate localization, in the minds of those clinicians whose experience with the disease has been greatest.

There are no pathognomonic signs of chronic interlobular pancreatitis, the suggestive factor being the association of various symptoms and physical signs, which have been sufficiently discussed. We do not consider the positive diagnosis of chronic interlobular pancreatitis possible, as we have never seen a case, diagnosed clinically chronic pancreatitis, operated upon and demonstrated to have this condition as the primary and most important lesion. It is true that the presence of pancreatitis either alone or in conjunction with other lesions may be strongly suspected, but the diagnosis cannot be made with the same degree of certainty as in gall-stones, duodenal ulcer or appendicitis. In a very few instances, none of which have come under our direct observation, marked emaciation showing some grave metabolic change, with intermittent constipation and diarrhea, and large stools with much undigested fat have pointed quite directly to the existence of a pronounced pancreatic lesion. In the vast majority of cases, however, these symptoms are not sufficiently marked to attract notice.

The differentiation from gall-stone disease is very difficult if not impossible in the greater number of patients. Fortunately treatment is the same and the patient does not suffer from the surgeon's inability to localize the various lesions causing the symptoms. The presence of pancreatitis may be considered highly probable if the patient has had symptoms referable to the bile-passages persisting over a long period.

In cases of chronic jaundice, the presence or absence of bile in the duodenal contents may aid greatly in the diagnosis. Even in advanced cases of chronic pancreatitis obstruction of the common duct is rarely absolute, consequently some bile escapes into the intestines. A stone in the common duct, unless it completely blocks the outlet, acts in a

similar manner. On the other hand, carcinoma of the head of the pancreas, as a rule, causes absolute obstruction and allows no bile to escape. Carcinoma of the bile-ducts, or of the gall-bladder causing secondary obstruction of the ducts may cause the same absolute occlusion and prevent the escape of the bile. Very often after the abdomen is opened and the pancreas examined directly by sight and touch, it is impossible to diagnosticate carcinoma of the head of the pancreas from chronic interstitial pancreatitis localized to the same region.

Chronic cholangeitis without gall-stones, and chronic appendicitis of the type manifesting itself chiefly or solely by upper abdominal symptoms, may also be mistaken occasionally for chronic pancreatitis.

Prognosis.—It does not appear that the existence of chronic pancreatitis as a complication of biliary tract disease materially increases the immediate mortality of operative treatment, unless the pancreatitis has passed beyond the curable stage. (See Tables at pp. 504, 505, 684.) Nor does it seem that the expectation of life is materially less. In other words, the prognosis, both immediate and remote, in early cases of chronic pancreatitis is much the same as in diseases of the biliary tract, and varies with the complications present in the gall-bladder and bile-ducts.

Treatment. *Medical Treatment.*—In mild cases where the diagnosis is extremely uncertain, medical treatment should be continued if improvement occurs. It is not improbable that many of the so-called "stomach complaints," catarrh, etc., are mild cases of pancreatic lymphangeitis that end in recovery. That these patients may be benefited by a by a "cure" at one of the famous springs is highly probable. But too great delay in resort to surgical measures is mistaken policy, especially when the diagnosis of the usual underlying biliary complaint is certain, since chronic pancreatitis, when once it has reached the stage of fibrous deposit, is incapable of being cured, although possibly the still further progress of the disease may be arrested by timely operation.

Surgical Treatment.—Surgical treatment of chronic pancreatitis aims at the accomplishment of three things:

1. Removal of the underlying cause.
2. Prevention of further involvement of the pancreas.
3. Cure of the pancreatic disease present.

Removal of the underlying cause is not always easy, as it is no easy matter to determine it in all instances. When, however, there is evidence of infection of the biliary system, meeting these conditions is sufficient to do away with the pancreatic disease in the majority of instances.

In cases of biliary infection, calculous or otherwise, operative treatment involves more or less prolonged drainage of the biliary tract, and this furnishes the correct mode of meeting the pancreatic condition.

The surest and most advantageous method of providing drainage and at the same time eliminating the main source of infection is by cholecystectomy. Drainage of the common duct should then be established.

There is no doubt that the pancreas may be drained through the opening in the common duct. In certain cases the discharge is peculiarly irritating to the skin, often causing excoriation. The presence of pancreatic ferment is demonstrated by the digestion of blood-serum and starch in alkaline solution. This we have done to prove that draining the common duct also drains the pancreas. These findings were obtained in cases where the patency of the outlet of the common duct was insured by the passage of a good-sized gall-stone explorer through the ampulla into the duodenum. When the pancreatic duct does not open into the sinus of Vater, this avenue for drainage of the pancreas is not open; but the operation is productive of good in that the primary focus of infection in the gall-bladder is abolished, and the accompanying lymphangeitis of the pancreas cured.

At times when the closure of the common duct is complete and likely to be lasting, a cholecystenterostomy may be best. Robson and Kehr are of the opinion that drainage by cholecystenterostomy will cure 97 per cent. of cases of chronic pancreatitis. Much as we dislike to disagree with such eminent authorities, we are still partial to external drainage. As a general rule this permits subsidence of the swelling of the pancreas and a re-establishment of the functions of the ducts. This drainage should be maintained for several weeks *at the very least.* If the gall-bladder, not the choledochus, has been drained it is often difficult to keep the fistula from closing too soon, but in cases of marked pancreatic disease the sinus may continue to discharge bile for months. This should not discourage either the patient or the surgeon, provided the presence of bile in the feces, even in slight amount, can be ascertained. Vautrin (1908) had to wait nine months in one case for the choledochus to become permanently patent and for the biliary fistula to close; Kehr, Körte and others have had to wait three or four months for the sinus to close. The prolonged drainage is beneficial; many patients have had recurrence of symptoms when the sinus closed and it has had to be reopened. It is our practice in cases of chronic pancreatitis, always to pass a sound through the common duct into the duodenum, to make certain that the passage is permeable. Only when

at operation the obstruction of the common duct is very marked, and the head of the pancreas very hard, do we think it proper to resort to cholecystenterostomy even as a secondary operation. Just how long the cholecysto-intestinal anastomosis remains patulous is a question, for in a few cases reoperation has demonstrated that the opening has closed. Cholecysto-duodenostomy is an operation having a higher mortality than cholecystectomy, which fact should be borne in mind when deciding the operation to be adopted in the individual case.

The problem of the treatment of pancreatitis when the gall-bladder seems to be practically normal is a more difficult one. In many of these cases close observation shows that the gall-bladder exhibits some interstitial thickening and possibly opacity of the serous coat. Such gall-bladders frequently contain thick tarry looking bile, many times containing micro-organisms, perhaps anaërobic. These are probably cases of mild hepatic infection, with inconspicuous involvement of the gall-bladder, or perhaps as suggested by Archibald's experiments are cases in which retrojection of nearly normal bile in small amounts occurs into the pancreatic duct. Drainage is as efficacious in these as in cases which show distinct pathological changes in the bile-tract. In some of these cases the origin of the trouble is in pyloric disease, and Finney's pyloroplasty, gastro-enterostomy, or other operation may be indicated. When no other lesion can be discovered Vautrin advocated attacking the pancreas directly; especially did he urge drainage of the retropancreatic tissues after exposing this region by mobilization of the duodenum (Fig. 164). In one case he uses the thermo-cautery to liberate the common duct from its position within the dense pancreatic head.

The means employed to remove the underlying cause of pancreatitis are also best adapted to prevent extension of the process and to cause a restitution to normal, so far as this is possible, of pathological lesions already present. In pancreatic lymphangeitis, proper drainage of the biliary tract reduces to a minimum the probability of an interstitial fibrotic change being superimposed upon it.

As we have already indicated, we think cholecysto-duodenostomy rarely or never is indicated for the treatment of chronic pancreatitis. If the common duct is patulous, it is extremely improbable, as indicated by Archibald's experiments, that this operation diverts the bile from its natural channel; and it is our belief that it should very rarely be employed as a primary operation. In those very rare cases of chronic pancreatitis where a biliary sinus persists indefinitely after drainage of the gall-bladder, cholecysto-duodenostomy may be employed

with propriety, and in most cases with complete relief of symptoms. But the danger of an ascending cholangeitis always exists, and the operation should not be adopted without mature consideration. If the gall-bladder has been inadvisedly removed at a previous operation, one of the other methods of restoring the continuity of the bile-passages must be employed (page 510).

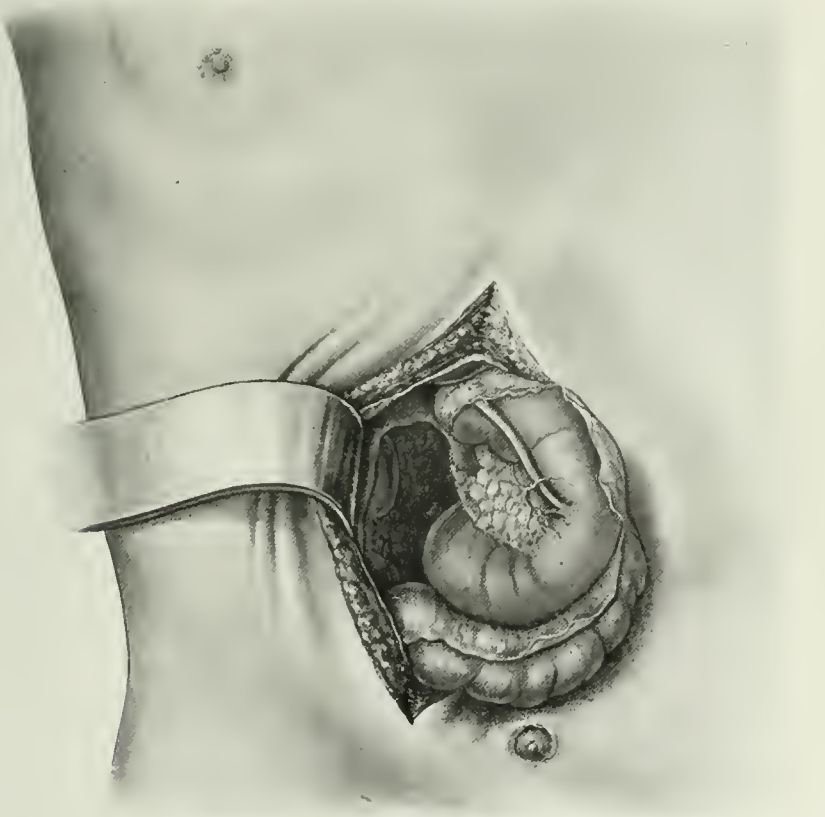


FIG. 164.—Exposure of the Head of the Pancreas and Common Bile-duct after Mobilization of the Duodenum. (*After Guibé.*)

The whole question of treatment for chronic pancreatitis resolves itself into an earnest endeavor promptly to treat upper abdominal disease by appropriate surgical means when medication has been ineffectual or when the symptoms point to a pathological condition not amenable to the ordinary therapeutic measures.

The following table contains a summary of the operations done by the senior author at the Lankenau Hospital during the past ten years:

OPERATIONS FOR CHRONIC PANCREATITIS (1910–1920)
(Lankenau Hospital)

Operation	Associated lesions	Cases	Deaths
Cholecystostomy	Carcinoma gallbladder	1	0
	Cholecystitis, chronic	21	1
	Calculus	16	0
	Pancreatic lymphangitis	3	0
	No other lesion	3	0
	Total	44	1
Cholecystostomy and marsupialization.	Cyst, pancreas, calculus	1	0
Cholecystostomy and choledochostomy.	Calculus	14	1
	Cholecystitis, chronic	2	0
	No other lesion	1	0
	Total	17	1
Cholecystectomy	Calculus	33	2
	Pancreatic lymphangitis	6	0
	Cholecystitis, chronic	5	1
	Cholecystitis, chronic, pancreatic lymphangitis	3	1
	Cholecystitis, chronic, cirrhosis liver	1	0
	Total	48	4
Cholecystectomy and choledochostomy.	Calculus	32	2
	Cholecystitis, chronic	2	0
	Total	34	2
Choledochsotomy	Calculus	8	1
	No other lesion	2	1
	Total	10	2
Cholecystoduodenostomy	Calculus	5	0
	Cyst, pancreas	1	0
	Cholecystitis, chronic	0	0
	Cirrhosis liver, ulcer duodenal, nephritis	1	1
	No other lesion	6	1
	Total	22	2
Hepaticoduodenostomy	No other lesion	1	0
Pancreatostomy	Calculus	1	0
Pancreatostomy and choledochostomy	Calculus	1	0
	Total	3	0
	TOTAL	179	12 (6.7%)

Pancreatic Calculi

In contradistinction to gall-stones, pancreatic calculi are of very infrequent occurrence. Johnston collected thirty-five cases in 1883, and by 1904 Lazarus found only a total of fifty-seven cases on record. Little that is definite is known of the factors which cause their formation. This aspect of the question, as well as certain aspects of the clinical pathology of the subject have been discussed at page 621.

Symptoms.—As is the case with all other chronic diseases of the upper abdomen, pancreatic calculi may exist for years without causing any definite symptoms or physical signs. Cases have been reported frequently where stones previously unsuspected have been found at autopsy. There is nothing distinctive in the group of symptoms usually associated with pancreatic calculi—pain, nausea and vomiting, jaundice, glycosuria, steatorrhea, azotorrhea and digestive disturbances.

Pain.—This varies in severity from an indefinite dull ache or sense of pressure, to attacks of acute colic scarcely to be distinguished from biliary colic. And just as "biliary colic" may be caused by violent peristaltic contractions of the gall-bladder and ducts, in the absence of all calculi; so it is probable that "pancreatic colic" does not always depend on the presence of calculi in the pancreatic ducts. In many cases of acute pancreatitis there is a history of previous attacks of epigastric pain, which may have been due to small hemorrhages or possibly to violent peristaltic contraction of the pancreas. In situation and radiation the pains usually are indistinguishable from those accompanying biliary colic, as is evidenced by the fact that pancreatic calculi have been so frequently diagnosed "gall-stones." The persistence of colic, after an operation for gall-stones, may be due to calculi in the pancreas (cases of Körte and Kümmell). This, however, is a very rare event. The pain is said to be more severe in the left epigastrium and to radiate to the left scapular region instead of the right; but in other cases the pain starts in the back and radiates around the side or straight through the body (Kinnicutt, 1902). *Nausea* and *vomiting* are constant accompaniments of the severe attacks of pain, as is the case in biliary colic. *Rigors* and *collapse* may also occur. Subsequent to these attacks of pain, calculi or fragments of calculi may be recovered from the feces. Analysis of these stones shows the usual composition of pancreatic calculi; as noted at page 622, they consist largely of calcium carbonate and phosphate. A stone that has been lodged long in the ampulla of Vater may become coated with bile-salts and biliary coloring matter until it resembles a gall-

stone, but the nucleus of the stone presents the characteristic composition. Such a stone may set up *jaundice* from obstruction of the common duct. This is not an uncommon symptom in connection with pancreatic lithiasis. It has the usual characteristics of obstructive jaundice. If the stone is passed jaundice is temporary, but if the stone lodges the jaundice remains and probably increases. Jaundice may be due not to blocking of the duct by a stone in the ampulla of Vater, but to an associated cholelithiasis or bile-duct infection, or to obstruction from pressure by the head of the pancreas, since in association with pancreatic lithiasis there always is a certain amount of pancreatitis. Occasionally this is of extreme degree; then there may be symptoms referable to the gastro-intestinal tract resulting from pancreatic indigestion, with steatorrhea and azotorrhea and other characteristic changes found in association with chronic interstitial pancreatitis.

Intermittent or permanent *glycosuria* occurs in about half of the cases, and is to be attributed to involvement of the islands of Langerhans in the sclerotic process. Alimentary glycosuria may be present occasionally. Flatulence, indigestion, loss of weight, etc., are to be referred to the accompanying pancreatitis rather than to the calculus.

Robson and Cammidge call attention to the fact that calcium oxalate crystals are present in the urine in over 40 per cent. of cases without jaundice, but in only 6 per cent. of jaundiced cases.

The most important characteristic about pancreatic calculus is that it is composed of material impenetrable to the X-rays and therefore can be demonstrated on an X-ray plate. If shadows are found by the X-ray, or if a stone is passed having the characteristics of pancreatic calculus, the diagnosis is simple. In other cases it is tentative and can be cleared up only by operation.

Diagnosis.—Diagnosis depends on X-ray examination. Gallstones are not often shown in a skiagram. The composition of pancreatic stones, if any are passed, is diagnostic, as they are composed of calcium salts without cholesterin or biliary coloring matter. Other than these two signs there is no method of arriving at a definite diagnosis. Lichtheim, in 1894, made a diagnosis of pancreatic calculi in a patient who suffered from epigastric colics, and later developed diabetes and characteristic diarrhea; and autopsy confirmed the diagnosis. Pepper, as long ago as 1882, made the diagnosis of pancreatic calculus in a patient under his care, but this was not confirmed by the passage of calculi, nor did the patient come to autopsy.

Kinnicutt (1902) collected seven cases in which the diagnosis was made during life, and Cipriani (1898) reported another case apparently overlooked by Kinnicutt. Glaessner (1913) made the diagnosis in four patients, two of whom passed calculi with the characteristics of pancreatic concretions; and Einhorn (1916) made the diagnosis in two patients, but in only one of these cases was the diagnosis confirmed by passage of a calculus.

Treatment.—Operation was suggested by Körte in 1898; it is the only rational method of treatment. The stone cannot be absorbed and while one may be passed occasionally, usually there are more left. As the condition is so often associated with chronic pancreatitis the latter also calls for operation, at which time the stone can be removed. Very often operation is undertaken because of a mistaken diagnosis of cholelithiasis or cholecystitis.

The treatment of colic is symptomatic as in gall-stones.

The *operation* consists in cutting down through the substance of the gland and removing the stone wherever it may be. In operation on the head of the pancreas it may be necessary to mobilize the duodenum to gain access to the affected area which may thus be approached from its dorsal surface (Fig. 164). In some cases the calculus or calculi may be exposed just to the left of the descending portion of the duodenum by an incision in the upper layer of the transverse meso-colon. If the stones are in the body or toward the tail of the pancreas, they are exposed best by division of the gastro-colic omentum (Fig. 160) or by the intercolo-epiploic route (Fig. 11). Cut surfaces of the pancreas unite after suture despite the presence of pancreatic juice on the surfaces of the wound; but in every case efficient drainage must be provided down to the site of the sutured area.

La Couture and Charbonnel (1914) collected 17 operations for stone in the pancreas including their own case: the results are known in 16 instances, 5 patients dying soon after operation (34 per cent. mortality), 10 surviving and being in good health when reported from 3 to 4 months later, and 1 dying of diabetes several years after operation. In Link's (1911) patient, constituting the seventh operation on record, a formal *pancreatostomy* was performed. Finding the pancreas even up to its tail filled with innumerable small stones, making it feel like a bag of fine sand, he exposed it through the transverse meso-colon. He next tore through the posterior parietal peritoneum covering the pancreas, seized the tail of the pancreas, and commenced its enucleation just as if it were a pyosalpinx covered with adhesions. He found it a comparatively easy task to free the gland as far to the patient's right as

the superior mesenteric artery. It was now possible to bring the tail of the pancreas out of the abdominal wound. Sponges were placed in its bed to arrest hemorrhage, which was not at all alarming; the pancreatic branches of the splenic artery were not ligated. After isolating the pancreas by gauze, it was split in the middle line for about two-thirds of its length. This opened the dilated duct of Wirsung, which was found to be filled with small facetted stones along its entire length. The stones, except those in the head of the gland, were removed;

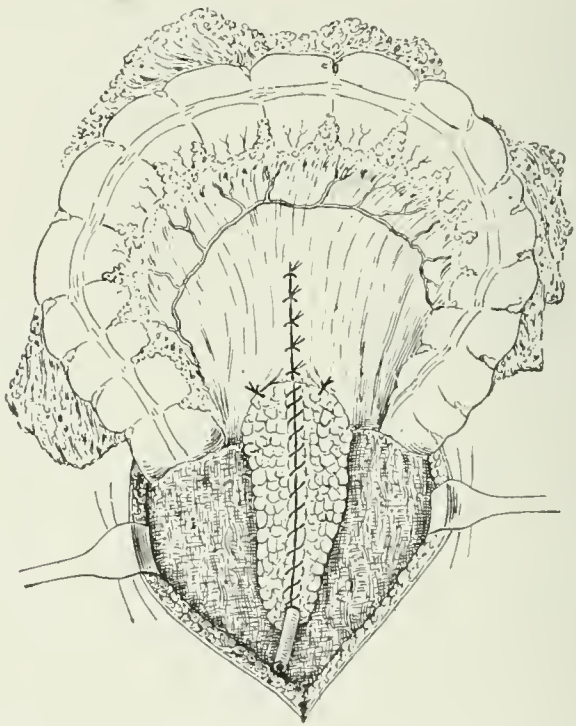


Fig. 165.—Pancreatostomy. (*Link.*)

and a drainage tube was laid in the dilated duct of Wirsung, projecting several inches from the tail of the pancreas. The gland was then closed around the tube with a continuous suture of No. 1 chromic catgut (Fig. 165). After stitching the opening in the meso-colon to the body of the pancreas, the great omentum was sutured over the sutured area of the pancreas. A gauze drain was placed beneath the pancreas, to protect it from the small intestines. Finally the abdominal wound was closed around the tail of the pancreas, which emerged at the lower angle of the incision. Pancreatic fluid was discharged freely at first

(about 750 cc. daily during the first week), but as the pancreatic inflammation subsided the secretions resumed their natural course into the duodenum. Several months after operation the drainage was slight, and the patient had resumed her usual active life.

In Dowd's patient (1915), not included in the statistics of La Couture and Charbonnel, recovery followed the evacuation of a pancreatic abscess, which contained 30 or more concretions. Though these concretions were composed chiefly of cholesterol and cholesterolesters, had practically no ash, and cast no X-ray shadows, Dowd considered them pancreatic in origin; and there was certainly nothing to suggest that they might be migrated biliary calculi. The patient continued in good health one year after the operation.

Injuries of the Pancreas

Uncomplicated injuries of the pancreas are very rare. This is to be explained by its deep situation, almost completely covered by other abdominal organs. Körte (1898) showed that in twenty out of thirty cadavers the pancreas was entirely covered by neighboring viscera. On account of its inaccessibility, rupture is less seldom uncomplicated than either gunshot or stab wound. Föwelin (1911) refers to twenty-nine cases of *isolated rupture* of the pancreas. The patient under his own care was the only case he could find of *uncomplicated stab wound;* the instrument entered from behind, to the left of the spine. Becker (1904) appears to have reported the only *uncomplicated gunshot wound* of the pancreas on record; the bullet passed to the left of the stomach through the gastro-splenic omentum. Both Föwelin's and Becker's patients recovered after prompt operation.

The *symptoms* of pancreatic injury are in no way characteristic, and an accurate diagnosis before opening the abdomen usually is impossible. Usually it is the complicating injuries (liver, stomach, etc.) which produce recognizable symptoms, and the lesion of the pancreas is discovered only incidentally. Wohlgemuth and Benczur pointed out (1910) that there is an increase in the amount of diastase in the blood and urine within a few hours after experimental or pathological obstruction of the pancreatic duct; and Noguchi (1912) suggested that chemical tests for diastase may be of value in determining whether or not the pancreas has been injured in cases of abdominal traumatism.

The *prognosis* depends upon the promptness of operation and very largely upon the presence of complicating injuries. Among twenty-nine cases of *isolated rupture* of the pancreas mentioned by Föwelin,

seven died without operation; twenty-two were operated on, with fourteen recoveries and eight deaths, a mortality of 36.3 per cent. Diehl (1911) collected twenty-two cases of *gunshot wounds* of the pancreas, only one of which was uncomplicated: among these patients six died without operation; sixteen were operated on, with nine recoveries and seven deaths a mortality of 43.7 per cent. *If the wound of the pancreas is not discovered at the time of operation, death nearly always is the result of overlooking it.* This was the cause of death in three out of the above seven fatal cases and doubtless contributed to the similar outcome in President McKinley's case. If operation is postponed, fat necrosis and retroperitoneal hemorrages may be found when the abdomen is opened; the case then will closely resemble one of acute pancreatitis.

Treatment.—Immediate laparotomy is indicated as in all injuries of the abdominal viscera. Usually the incision is made in or near the median line, for purposes of exploration. In every case in which there is any possibility, even remote, of pancreatic injury, the pancreas should be explored, either through the gastro-colic or the gastro-hepatic omentum. The former is the preferable route, as giving better exposure. As directed at page 295 the incision in the gastro-colic omentum should be *at least 8 cm. long*, and should pass below the gastro-epiploic vessels (Fig. 160, p. 651). Bleeding from the pancreas should be checked by gauze packing, unless the point is freely accessible when suture may be attempted. But even where the injury has been securely sutured, the surgeon should not neglect to drain the injured region. Should a pancreatic fistula develop, the discharge may be much lessened and healing accelerated by putting the patient on Wohlgemuth's antidiabetic diet (page 658).

CHAPTER XXII

TUMORS OF THE PANCREAS

Solid Tumors of the Pancreas

These include **carcinoma, sarcoma** and **adenoma,** of which carcinoma is the most common.

Carcinoma of the Pancreas.—Various statistics have been reported indicating the frequency of carcinoma of the pancreas. But figures derived from records published before 1900 are not entirely trustworthy, as until the general recognition of chronic pancreatitis which followed Mayo Robson's researches, many cases of the latter disease were regarded as carcinomatous. Without histological examination it is difficult to distinguish one from the other. With these limitations in mind, the following classical statistics may be quoted: among 33,788 cases of carcinoma in males Bashford found *primary carcinoma* of the pancreas in 526; and among 50,660 cases in females, the tumor was *primary in the pancreas* in 474. This shows a distinctly greater frequency of primary carcinoma in the male sex.

Secondary carcinoma of the pancreas is of little interest to the surgeon. It seldom is the result of metastasis; in almost all cases it is due to extension from neighboring organs, particularly the stomach. Secondary carcinoma has been considered much more common than the primary form, only two of Eppinger's nineteen cases being primary. But Hale White's statistics (1897) do not uphold these figures, and Ferguson (1910) says that primary carcinoma of the pancreas is more common than secondary. If Oser's statement (1903) is true that 10 per cent. of all cases of primary carcinoma of the stomach involve the pancreas secondarily, there can be no doubt that secondary growths are more common than primary.

Age.—Carcinoma of the pancreas is a disease of middle or advanced life. In nearly all reported cases the patient has been over forty years of age. Cases occurring in childhood have been reported by Bohn, Kuhn, Simon and Dutil. Their cases were respectively seven months, two years, thirteen years and fourteen years of age.

Sex.—As indicated already, carcinoma of the pancreas is more common in men than women, the relation being about three to two, much the same ratio which obtains in cases of chronic pancreatitis.

Mirallié (1893) reported 100 cases of primary pancreatic cancer, sixty-nine being in men and thirty-seven in women. Oser (1903), including Mirallié's cases, reported 246 in men to 142 in women. Among the sixteen cases of primary carcinoma arising in the body of the pancreas, studied by Leriche (1910), eleven occurred in males, and only five in females.

Pathology.—While diffuse growths occasionally occur, primary carcinoma usually is of small size. This fact explains why primary involvement of the head of the pancreas with metastasis to the liver is so often mistaken for primary carcinoma of the liver, the original growth in the pancreas being overlooked on account of its small bulk. When a pancreatic cancer grows to a large size, invades neighboring structures and forms extensive adhesions, the starting point of the growth is not easily determined.

In primary carcinoma the head of the gland is the most frequent site of growth, although the body, tail or whole gland may be involved. Oser in sixty-eight cases found the following distribution: head, thirty-nine; whole organ, nineteen; tail, four; head and body, three; body and tail, one; head and tail, one. In Mirallié's 106 cases the head was the site of the growth in eighty-two. In Segré's cases (1888) the distribution was as follows: head, thirty-five; entire gland, nineteen; body, two; tail, one. Robson and Cammidge give the following percentages: head 62 per cent.; tail 5.5 per cent.; body 3.5 per cent.; diffuse growth 29 per cent.

The point of origin of the growth usually determines its type (Hulst 1905). If it arises in the epithelium of the excretory ducts it develops into a cylindrical celled tumor of the adenocarcinomatous form. Letulle contends that a carcinoma springing from the ducts of Wirsung is composed of solid alveoli of spheroidal and not cylindrical cells. Olivier (1894) reported a case in which the cells of the tumor apparently arose directly from the ducts and the growth was of the spheroidal-celled solid alveolar type without the central lumen seen in adenocarcinoma. Hulst thinks that these spheroidal-celled tumors with solid alveoli take their origin from the glandular epithelium.

Whatever the origin of these two forms there usually is developed sufficient fibrous tissue to make them hard and firm. The great majority of pancreatic carcinomata are of the *scirrhous variety*. Occasionally the growths are very cellular, forming a tumor of *encephaloid type*. Columnar-celled tumors may undergo *colloid degeneration*, several having been reported.

No connection has ever been definitely determined between the

islands of Langerhans and the origin of carcinoma. Hillier and Goodall (1904) described a form of carcinoma supposed to arise from the islands of Langerhans. These differ from the other forms in the great irregularity of their cells. Olivier believes that some of the so-called cases of primary carcinoma of the pancreas arise in the duodenum from the glands of Brunner.

Sooner or later the growth of a carcinoma of the pancreas encroaches on one of the excretory ducts and causes complete obliteration of its lumen. This results in the development of a chronic interstitial pancreatitis in the area drained by the affected duct. Moreover, the tumor itself, as already noted, is surrounded by a reactionary growth of connective tissue.

Carcinomatous degeneration in chronic interstitial pancreatitis is spoken of as a possible complication by Hulst, but has not yet been proved to occur.

Neighboring organs may be involved from primary carcinoma of the pancreas by metastasis, extension, or the formation of adhesions.

Metastasis occurs in the liver as a rule. When it is extensive the determination of the site of the primary growth is extremely difficult. This applies particularly to cases of carcinomatosis said to arise from primary tumors of the pancreas.

The position, size, and direction of the growth of the tumor determine the organs involved by extension or adhesions. Carcinoma of the head of the pancreas encroaches on the common bile-duct, and causes steadily increasing obstruction until the lumen is completely obliterated. This blocking of the common duct is evidenced by progressively deepening jaundice, which usually is associated with enlargement of the gall-bladder, unless previous gall-stone disease has caused thickening and shrinkage from fibrous change. The stomach and duodenum from their relations to the pancreas are naturally the areas to suffer most frequently. Obstruction to the duodenum or pylorus causing dilatation of the stomach, ulceration into the duodenum or stomach, compression of the transverse colon or stomach and of various large vessels in the abdominal cavity, and even compression of the left ureter causing hydronephrosis, have all been described as resulting from carcinoma of the pancreas.

Glycosuria is not a frequent complication but when it does occur it indicates involvement of the islands of Langerhans, either in the chronic pancreatitis resulting from the growth or in the tumor itself.

Symptoms.—In carcinoma of the pancreas there are no pathognomonic symptoms. Following the original clinical picture drawn

by Bard and Pic (1888), it was considered for a long time that steadily increasing jaundice, enlargement of the gall-bladder and rapid emaciation constituted a characteristic syndrome or group of symptoms, which indicated carcinoma of the head of the pancreas. Now, however, it is generally recognized that this syndrome may result from any condition which causes obstruction at the papilla of Vater. In carcinoma of the pancreas it is only when the growth is in the head of the gland that this obstruction results, so there are necessarily cases that do not exhibit the so-called characteristic symptoms. On the other hand various conditions, such as carcinoma of the bile-ducts, chronic interstitial pancreatitis, carcinoma of the gall-bladder, etc., may cause obstruction of the choledochus and of the duct of Wirsung, and thus give rise to this same group of symptoms. A diagnosis of carcinoma of the pancreas, therefore, must be made with a certain degree of reserve; and only after careful consideration of the history, physical signs and laboratory findings in each individual case.

Jaundice.—In carcinoma of the head of the pancreas the common duct is steadily compressed and eventually occluded by the growth, jaundice resulting. The characteristic feature of the jaundice is steady progress without intermission or remission, differing in this particular from that caused by a stone in the duct, which is subject to variation, as obstruction is seldom so complete as to prevent absolutely the passage of bile. Complete obstruction is shown by the persistent absence of bile and pancreatic ferments in the duodenal contents. Occlusion of the common duct by cancer of the choledochus, or of the papilla of Vater, or by extension from cancer of the gall-bladder or pylorus gives rise to the same type of jaundice, and it is indistinguishable from that caused by carcinoma of the head of the pancreas.

The manufacture of bile continues in spite of the obstruction and the jaundice gradually deepens until it becomes the "black jaundice" of older writers.

If the body or tail of the pancreas is affected the disease may run its whole course without causing jaundice. As a symptom of carcinoma of the pancreas, jaundice is peculiar to growths involving the head of the gland, the only position in which the tumor can compress the common duct.

Extension to the head of the pancreas or metastasis to the liver or the lymph-nodes in the gastro-hepatic omentum is necessary before jaundice will arise in cases of carcinoma of the body or tail of the pancreas.

Enlargement of the Gall-bladder.—If carcinoma is the primary condition and there has been no previous gall-stone disease or duct inflammation; in other words, if at the time of onset of obstructive jaundice the gall-bladder is normal, back pressure on the ducts causes distention of the gall-bladder from the accumulation of bile. As the obstruction increases and the pressure becomes greater, no more bile can reach the gall-bladder and the continuing distention is due to secretion of mucus.

If there has been previous inflammation from stones or non-calculous infection the deposit of fibrous tissue in and around the walls of the gall-bladder interferes with its elasticity and it is consequently incapable of distention. In these cases the gall-bladder is small and contracted irrespective of the amount of back pressure.

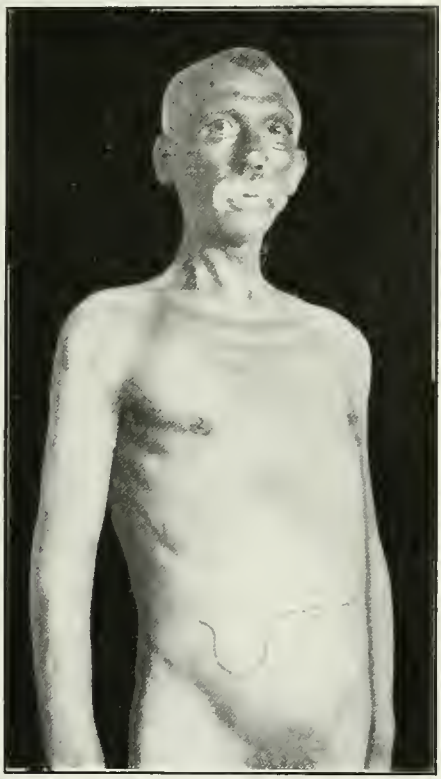


FIG. 166.—Obstructive Jaundice with Enlargement of the Liver and Gall-bladder. Presumably from Carcinoma of the Head of the Pancreas. An Inoperable Case. From a Patient in the Lankenau Hospital.

Pain.—Opinions differ in regard to the occurrence and the degree of pain. Opie says, "Pain is one of the earliest and most common symptoms." On the other hand Robson and Cammidge say, "Pain is usually absent or unimportant;" while Robson and Moynihan say, "In one-half the cases at least, the suffering is exquisite." Chauffard (1908) called particular attention to very severe crises of abdominal pain, which he described as a veritable visceralgia. He described the patients as sitting with flaccid abdomen, bending forward to their knees, and unable to eat anything. From these contradictory statements it may be inferred that all varieties in character and severity of pain occur; and that, while pain may be a conspicuous feature, it is not a necessary factor in the clinical picture of a case of pancreatic carcinoma. In cases which have come under our own observation, pain, while seldom

a prominent symptom, has rarely been entirely absent. Various explanations of the cause of the pain have been given. Continuous pain increasing in severity until death is ascribed to pressure on the celiac ganglia. Colicky pain is supposed to be due to obstruction of the duct of Wirsung or the common duct setting up attacks of true colic. When the head of the pancreas is involved, the usual seat of pain is in the epigastrium or right hypochondrium, from which situations it may radiate to the back or shoulder. When the growth originates in the body of the gland, Leriche (1910) says the earliest pain is in the left hypochondrium.

Loss of Weight and Strength.—Early in the course of the disease the loss of weight and strength is very striking, and this continues progressively and rapidly until the patient dies from exhaustion. Death seldom is delayed more than a few months after the appearance of jaundice. Pancreatic cancer is said to be the most rapidly fatal of all forms of malignant disease. The entire duration of the malady, from the recognition of the first symptoms, seldom exceeds a year.

Digestive Disturbances.—These are of two kinds: (1) those due to obstruction of the duodenum or pylorus, causing symptoms of stricture; and (2) those due to interference with pancreatic digestion. The former give the usual picture of pyloric obstruction, anorexia, distress and distention after eating, and delayed vomiting, together with the well-known physical signs and laboratory findings of gastric dilatation from obstruction.

If the duct of Wirsung is occluded to such an extent that little or no pancreatic juice reaches the intestine, the usual evidences of *pancreatic insufficiency* are found. There is early loss of weight and strength much greater than that usually caused by the growth of a malignant tumor.

The changes that occur in the feces when pancreatic juice does not reach the intestine have been described at page 626. Briefly, they are increase in the amount of unabsorbed fat in the feces from the normal of 5 per cent. to 50 per cent. or more (in cancer of the head of the pancreas the unabsorbed fat in the feces may amount to 90 per cent. of that ingested); diminution in the percentage of split fat from the normal of 70–80 per cent. to as low as 15 per cent. Even in those cases in which there is no increase above normal in the actual amount of fecal fat, there is still a diminution in the proportion of split fats to 40 per cent. or less. These findings and their significance have been discussed in relation to diagnosis (page 627).

Tumor.—Tenderness is unusual, but may occasionally be present and interfere with palpation. In the majority of cases no mass referable to the pancreas is palpable, the tumor usually being small and confined to the head of the gland. While it is true that the greater number of malignant growths of the pancreas are fixed, this is not a universal rule by any means. Although the pancreas is retroperitoneal, it is invested by loose areolar tissue which permits considerable motion, and unless it is anchored by inflammatory or malignant adhesions, mobility is marked. Mobility of a tumor, therefore, does not necessarily rule out a pancreatic origin. Body and tail tumors are more likely to be movable than those arising in the head of the gland. As a rule, however, malignant growths are either too small to be felt or are firmly fixed by adhesions.

In thin patients with relaxed abdominal walls any considerable enlargement of the pancreas can be detected, but it takes a comparatively large tumor to be recognized as such. When palpable it usually transmits pulsation from the aorta; and if, as in Poncet's case(1890) there is also a murmur the lesion may be mistaken for an aortic aneurysm. In one of Giovanni's patients (1916), both of whom presented this symptom, aneurysm of the celiac axis was present. Mirallié (1893) reported that there was a palpable tumor in one-fourth to one-fifth of his 113 cases. A tumor was palpable in nine out of fourteen cases of carcinoma of the body or tail, analyzed by Leriche (1910); usually the mass presented through the gastro-hepatic omentum, displacing the stomach downward. Although the pancreas itself usually cannot be palpated there often is an epigastric tumor due to the distended gall-bladder. In rare instances two tumors have been felt, one the pancreatic growth and the other the distended gall-bladder. An enlarged gall-bladder is easily felt as a rounded, smooth tumor which is just beneath the abdominal wall and moves with respiration. In fifty eight out of sixty-two cases of obstruction of the common bile-duct due to carcinoma of the pancreas reported by Ecklin (1896) there was recognizable enlargement of the gall-bladder.

The liver has no distinctive features, its size varying from normal to marked enlargement, depending upon the degree and character of the secondary involvement. Considerable increase in the size of the liver is to be expected in the late stages of the disease (Fig. 166).

Glycosuria occurs in a certain proportion of cases. It appears when the growth has advanced to such an extent that it has destroyed the greater number of the islands of Langerhans; as is the case in chronic interlobular pancreatitis, the islands of Langerhans persist

for some time after the parenchyma has been destroyed, so that the appearance of sugar in the urine usually is a late development if it occurs at all. Mirallié (1893) reported glycosuria present in thirteen of fifty cases. Both alimentary glycosuria and transient glycosuria have been reported, but the mere presence of sugar in the urine does not serve to differentiate the form of pancreatic disease causing it.

In cases of continued jaundice there always is a *tendency to hemorrhage*. In cancer of the head of the pancreas, gastric, intestinal, subcutaneous, and even nasal and oral hemorrhages are not uncommon when hepatic involvement has produced portal obstruction. Spontaneous hemorrhage usually is not serious, but that which occurs after operation and which is due to changes in the blood, is distinctly dangerous. Persistent and uncontrollable postoperative oozing frequently is fatal even in those cases where nothing but an exploratory incision has been undertaken.

The analysis of *stomach contents* is comparatively of little value in differentiating pancreatic from other upper abdominal growths, but in doubtful cases useful confirmatory evidence is occasionally obtained by analysis of the *duodenal contents*.

Left-sided hydronephrosis, ascites, edema of the legs and enlargement of the spleen sometimes are present as pressure symptoms. Another pressure symptom which was mentioned at page 697 is aortic pulsation with a murmur. Chylous ascites has been reported; it is due to rupture of the thoracic duct.

Elevation of temperature results only when an intercurrent infection arises.

The **clinical course** of a case of carcinoma of the pancreas is remarkable for its rapid progress to a fatal termination. The interference with pancreatic digestion, the jaundice, and the cachexia from a malignant tumor combine to make this one of the most rapidly fatal of malignant diseases. Death usually ensues within six to eight months after the appearance of jaundice but it may be delayed for two or even four years after the onset of the earliest symptoms.

Diagnosis.—In a typical case of primary carcinoma of the head of the pancreas the diagnosis can be made with a fair degree of certainty, but unfortunately not at an early period of the malady unless the growth arises very close to the main excretory duct and rapidly invades the papilla of Vater or compresses the common bile duct. The patient, usually over forty years old, complains for a vague period of upper abdominal symptoms having no localizing character. After a longer or shorter time jaundice appears and

painlessly and continuously deepens; the gall-bladder enlarges and the patient loses weight and strength very rapidly. Pain may or may not be a conspicuous feature. The persistent absence of bile and pancreatic juice from the duodenum is a valuable confirmatory sign, and indicates complete obstruction. The feces may show signs characteristic of failure of pancreatic digestion due to the absence of pancreatic juice. A tumor connected with the pancreas seldom can be recognized. When there is such a tumor its relations may be determined by inflating the stomach and colon. Theoretically this should completely obscure a tumor of the pancreas, but from the number of cases reported where operation was undertaken for other conditions, it is evident that pancreatic tumors often exhibit characteristics common to various abdominal tumors. The size of the liver is of comparatively little value in differential diagnosis.

The main factors on which a diagnosis of cancer of the head of the pancreas is based are progressive jaundice, enlargement of the gall-bladder, rapid loss of weight and strength, and persistent absence of bile and pancreatic juice from the duodenum. Such a clinical picture indicates complete obstruction of the common duct, and carcinoma of the head of the pancreas is the commonest cause of such obstruction; yet other causes cannot always be excluded, especially carcinoma of the common bile-duct or the papilla of Vater causing coincident obstruction of the duct of Wirsung. Carcinoma of the hepaticus causes obstructive jaundice but there is no pancreatic insufficiency; and even when the duct of Wirsung is obstructed by a growth, pancreatic insufficiency may not develop if the accessory duct of Santorini is patulopus.

Atypical cases of carcinoma of the pancreas which develop after symptoms of gall-stones, gastric or duodenal disease, as well as those in which obstruction of the choledochus does not occur, and those exhibiting a palpable, movable tumor, cause extreme difficulty in diagnosis and it is at times impossible to make a positive diagnosis.

Carcinoma in the body or tail of the pancreas can scarcely be differentiated from other palpable tumors in the upper abdomen, for it may be as freely movable as any of the commoner tumors, and will not cause obstructive jaundice nor symptoms of pancreatic insufficiency. Gastric and intestinal symptoms and the more superficial position of the tumor favor the diagnosis of an extra-pancreatic origin, but much reliance cannot be placed on these points.

Differential Diagnosis. *Carcinoma of the Common Duct.*—For practical purposes this includes carcinoma of the papilla, of the

ampulla of Vater, of the common bile-duct and of the main hepatic duct.

Primary carcinoma of the common bile-duct is very much less frequent than carcinoma of the pancreas. Contrary to what might be expected, gall-stone disease does not appear to be a predisposing factor in cancer of the bile-passages.

In primary carcinoma of the common duct the onset usually is insidious and after a varying period in which ill-defined epigastric symptoms are manifested, jaundice appears and deepens gradually but persistently. So far the resemblance to pancreatic carcinoma is striking, but until the pancreatic ducts are obstructed by the growth in the choledochus there are no signs of pancreatic insufficiency. From this time on the case runs the same clinical course as carcinoma of the head of the pancreas and the two conditions are, as a rule, impossible of differentiation, except on the grounds that carcinoma of the head of the pancreas is much commoner than common duct cancer and is associated with more rapid emaciation and loss of strength because of the early interference with pancreatic digestion.

Carcinoma of the gall-bladder simulates cancer of the head of the pancreas only when the growth has extended to and occluded the ampulla of Vater, causing both obstructive jaundice and pancreatic insufficiency. When jaundice is due to involvement of the liver, and not to obstruction of the choledochus, bile is not absent from the duodenum.

The history of gall-stone disease or of prolonged upper abdominal symptoms, the comparatively late onset of jaundice, the absence of the smooth, rounded, gall-bladder tumor and in its place a hard, nodular tumor or no tumor at all, make a clinical picture much different from that of carcinoma of the head of the pancreas.

Extension of malignant disease to the ducts from neighboring organs is preceded by symptoms of the primary disease, and jaundice is a late symptom.

Stone in the Common Duct.—The history of previous attacks, the onset of jaundice after colic, the intermittent character of the jaundice, the fact that there is little or no loss of weight and strength for a long time after the initial symptoms, the absence of a gall-bladder tumor and the presence of bile in the duodenum all serve to differentiate gall-stones from pancreatic cancer. In addition, obstruction of the common duct by a stone usually gives rise to intermittent fever, while the temperature is normal or subnormal in cancer of the pancreas.

Chronic Interstitial Pancreatitis.—When the abdomen is opened and the pancreas examined directly by sight and touch it often is impossible to differentiate carcinoma and pancreatitis. A preceding history of gall-stone disease or other predisposing factor to pancreatitis, and, as a rule, the slower loss of weight and strength in pancreatitis help in differentiation. In inflammatory disease the gall-bladder usually is contracted, but even when it is distended it is sometimes possible to distinguish it from the distended gall-bladder which results from malignant obstruction by its friability. In malignant disease the gall-bladder, unless itself involved in the carcinomatous process, is much more resistant and sutures inserted in it are much less apt to tear out than in the case of a gall-bladder which is the seat of longstanding inflammatory change.

In undetermined cases the patient should be given the benefit of the doubt and treated as for pancreatitis.

Cancer of the Liver.—Absence of the evidence of complete obstruction of the bile-ducts, the late onset of jaundice, enlargement of the liver, palpable nodules on the surface of the liver, and lack of symptoms and signs due to interference with pancreatic digestion indicate the probable diagnosis.

Cancer of the Pylorus.—Even if jaundice does occur it is a late symptom due to extension or metastasis. The gastric symptoms predominate although pyloric and pancreatic cancer may occur apparently at the same time and the primary seat of the tumor be uncertain.

The *gastric crises of tabes* may be distinguished from the visceralgia which characterizes some cases of pancreatic carcinoma by attention to other signs of locomotor ataxia.

Treatment.—Medical treatment is symptomatic.

In cases of doubt abdominal section is indicated as the condition may be amenable to operative treatment. Confluent enlarged lymph-nodes point to carcinoma while discrete glandular enlargement usually indicates pancreatitis. In nearly every case of carcinoma of the pancreas the disease has progressed too far to permit removal. Complete removal of the pancreas appears to be necessarily fatal. The case attributed to Billroth (1884) of recovery after complete extirpation of the pancreas, is rejected by Sauvé (1908), who says it is cited everywhere but nowhere reported. In cancer of the body and tail a number of successful removals have been recorded. In 1910 Finney collected six resections of the body or tail of the pancreas with two deaths from the operation, two survivals for a few months, one reported as "recovered" but with no after history, and Finney's own patient (cystaden-

oma) who was in good health sixteen months after operation. In excisions of the tail, the stump may be closed, and any fistula may be expected to close under antidiabetic diet. In resections of the body, the head and tail should be sutured together, as in Finney's own case.

Compared to the number of actual cases, successful removal must be looked upon as only rarely possible, but should always be undertaken if there is the slightest possible chance of success. Sauvé quotes Terrier's maxim: *Dans le doute, ne t'abstiens jamais.*

A *radical operation* for carcinoma of the *head of the pancreas* involves also removal of the second or descending portion of the duodenum. The operative technique has been well systematized by Desjardins (1907) and by Sauvé (1908), the latter of whom uses the term cephalic duodeno-pancreatectomy to describe the operation. He collected sixteen cases of cephalic pancreatectomy of which the details are known, and refers to five others merely mentioned in journals. Of these sixteen patients, nine survived the operation (Ruggi, Sendler, Codivilla, Biondi, Tricomi, Franke, Duval, Villareal, Mauclaire) and three patients were in good health a year later, but *these three did not have cancer* (Sendler, Biondi, Duval). Of all the operations collected by Sauvé that of Codivilla was the most ideally complete: cephalic duodeno-pancreatectomy, with gastro-jejunostomy in-Y, and cholecystenterostomy; his patient lived twenty-four days, and succumbed then to the preexistent cachexia. The patients of Tricomi and Franke survived for five months and six months, respectively. Cordoy, Mauclaire, Villaréal, and Moynihan employed duodeno-pancreatectomy; while Cunéo adopted the method employed by Tuffier in a case of carcinoma of the papilla of Vater invading the pancreas. This consisted in enucleation of the head of the pancreas and excision of the ampulla of Vater; the stump of the pancreas was fixed in the abdominal wound. Even in the technique recommended by Sauvé, and which is described in detail at page 795, the stump of the pancreas is fixed in the abdominal wound. In the technique proposed by Desjardins the pancreatic stump is implanted into the intestine. Coffey (1909), in experimental work on dogs, sought a technique to facilitate such a step. The best way to do this, he found, was to throw the lumen of two intestines into one by using a loop, and thus allow ample room for invagination of the pancreas (page 799).

Cholecystostomy and cholecystenterostomy do not prolong life to any extent and a number of the patients die as a result of the operation. Symptomatic relief usually is afforded to those who survive: the jaundice lessens or disappears, digestion and appetite improve,

and the patients enjoy an interval of reasonable comfort which would be absent were operation refused. The results of these operations in cases of malignant disease have been noted at pages 505 and 591. The immediate results in patients under the senior author's care during the past eight years are indicated in the accompanying table:

OPERATIONS FOR CARCINOMA OF PANCREAS (1912–1920)
(Lankenau Hospital)

Operation	Associated lesions	Cases	Deaths
Cholecysostomy		1	0
Cholecystectomy	Calculus	3	3
Cholecystectomy and choledochostomy	Calculus	1	1
Cholecystenterostomy		5	1
		10	5

Sarcoma of the Pancreas.—Kakels in 1902 collected twenty-one cases. Primary sarcoma is very rare and while secondary involvement occurs more frequently it is still an uncommon condition. In 1909 Ravenna recorded from the literature twenty cases of primary sarcoma and reported two original cases. Villard and Stéfani have since recorded an operation in which marsupialization was done for polycystic sarcoma of the pancreas; death occurred eight days later.

Primary sarcoma is of the small round-celled variety, or lymphosarcoma. *Secondary* sarcoma is also usually lymphosarcoma and arises from the mediastinal or abdominal lymph-nodes or from the duodenum. A number of cases of melanotic sarcoma are reported and usually have resulted from metastasis from the eye.

Segré (1888) reported two primary sarcomas occurring in 11,492 autopsies. Hale White (1897) found one in 6708 autopsies. In Finney's report (1910) of seventeen cases of resection of the pancreas, sarcoma occurred four times, two of the patients recovering. One of these died of recurrence within a few months.

Diagnosis is scarcely possible before operation.

If the tumor is in the body or tail successful removal is possible. If the growth is in the head of the gland cephalic duodeno-pancreatectomy may be done but usually patients are seen too late for radical treatment to be successful.

Adenoma is of pathological rather than surgical interest (Cecil, 1911). The diagnosis is made by microscopic examination and there

are no characteristic symptoms or physical signs. A certain number of successful removals have been reported.

Cysts of the Pancreas

The percentage incidence of pancreatic cysts is difficult to determine because the majority of collected cases are reports of operation, most often only incision and drainage, where the true origin and connections of the tumor could not be ascertained with any degree of accuracy. For this reason it is probable that many cases are classed as pancreatic although the cysts have arisen independently of the pancreas.

Classification.—The simplest and most convenient classification is into true cysts and false or pseudocysts. The former include those due to retention of pancreatic secretion, cystic new growths, hydatid cysts and congenital cystic disease. Pseudocysts arise in close association with the pancreas and involve it secondarily. These cysts usually are formed by effusions the result of abdominal injuries. Some authors classify with these extra-pancreatic pseudocysts those which arise in the pancreas as a result of hemorrhage from injury or acute pancreatitis. As these are practically indistinguishable from true retention cysts such a classification is not rational from a clinical standpoint.

Etiology. *Age.*—The years between twenty and forty furnish most of the cases. Railton (1896) reported a pancreatic cyst in a six months old child; Shattuck (cited by Richardson, 1882), one in a child thirteen months old; Connelly (1911) and Richardson (1895), each one in a child fourteen months old; while Stieda (1893) reported a case in a man seventy-six years old.

Sex.—Robson and Cammidge (1907) say that true cysts are more common in women and pseudocysts more common in men, presumably because the latter are more exposed to injury. Körte's statistics (1898) show almost equal incidence in men and women.

Traumatism.—Experimental evidence shows that injury to the pancreas causing hematoma results in cyst formation.

Pancreatic Cyst Following Trauma; External Pancreatic Fistula Spontaneously Developed; Operation; Recovery

A woman 27 years of age came under the care of the senior author in the Lankenau Hospital. Her *chief complaint* was a fistula in the left hypochondriac region. *Five years previous* to her admission she had suffered from dyspepsia for a period of 6 months, and

since that illness a dull pain had persisted to the left of the epigastrium. *Three years ago* she fell and struck her lower left chest; shortly afterward she noticed a lump in the region where the pain had previously been felt. This lump had grown slowly for the past two years, causing a sensation of weight and fulness in the upper left abdomen. *Two days ago* this lump ruptured externally, and large quantities of white cloudy fluid were discharged, with some relief to the discomfort.

The fistula, which was 3 cm. below the left costal margin, exuded clear mucus which was very irritating to the skin; and the skin was excoriated over an area 5 cm. in diameter. Examination of the fluid showed that it partly digested the white of an egg.

At operation the fistula was traced through the gastro-hepatic omentum to the pancreas, where the tract was ligated and the fistulous tract excised. Recovery was uneventful.

In thirty-three (28 per cent.) of the 117 cases collected by Körte there was a history of some abdominal injury, these injuries usually being direct blows of varying degrees of violence; although falls, compression of the abdomen, etc., have also preceded and apparently caused a certain number of pancreatic cysts.

Previous Pathological Conditions.—All conditions likely to be associated with chronic interstitial pancreatitis have been described in connection with pancreatic cysts. Calculi, tumors, chronic infection of the duodenum and bile-passages, duodenal and gastric ulcer and pancreatic lymphangeitis have been mentioned as pathological lesions accompanying pancreatic cysts. Whether they cause both the cyst and the pancreatitis or whether the cysts result from the pancreatitis is hard to determine. Robson and Cammidge favor the latter supposition and think that when cyst, calculus and chronic pancreatitis occur together the two former have a common cause in the latter.

Acute hemorrhagic pancreatitis has frequently resulted in cyst formation.

Pathology. *Retention Cysts.*—It is doubtful if simple obstruction of the pancreatic duct or one of its branches leads to the formation of a cyst or cysts. Those observers who think that it does believe that there is a change in the character of the pancreatic juice, which as a result of stagnation becomes less easily absorbed. Subsequently this causes disintegration of the glandular tissue with the formation of a cavity into which secretion and exudate are poured out. Failure to cause cyst formation in certain cases is ascribed to nonclosure of the duct of Santorini. After causing only slight dilatation of the duct Senn (1885) suggested that complete obstruction would lead to atrophy, but that intermittent obstruction, as by a calculus, would cause cyst formation in the same manner that a stone in the ureter causes hydronephrosis. While such theories may furnish a partial explanation of the mode of origin of cysts, there are other unknown

factors, as is shown by the fact that the same etiological factors blamed for the formation of cysts are brought forward as direct causes of chronic interstitial pancreatitis. We have no knowledge as to why the same conditions at one time cause cyst formation and at another do not. There is always a certain amount of pancreatitis in association with cysts of the pancreas, and Robson and Cammidge express the opinion that the cyst is the direct result of the compression of the duct by contracting bands of scar tissue. This removes the difficulty only a step farther, as it does not explain why a cyst occurs in one case of pancreatitis and not in another.

Thiroloix (1897) altered the contents of the ducts before ligating both, by injecting soot and carbolized vaseline, and caused the formation of a cyst with, in addition, very advanced chronic pancreatitis. The determining factor of cyst formation in this case is supposed to have been the alteration of the pancreatic juice by the foreign matter injected, which interfered with absorption.

It is not yet clear that the chronic pancreatitis may not be the result of extension of the chronic inflammation always found around the wall of a cyst.

Pancreatic calculi, gall-stones, duodenal ulcer, tumors and lymphangeitis have all been found in association with retention cysts but their connection therewith is not understood. If they obstruct the duct this may cause a rapid increase in size of a preformed cyst, but there is no definite reason for saying that they are causative factors.

All sizes and conditions of cysts occur; uni- and multi-locular cysts, single and multiple, large and small, occur in all sorts of combinations and the etiology of the various combinations is uncertain.

Virchow (1887) described a multiple dilatation of the duct of Wirsung, caused by obstruction from a tumor of the duodenum, which he termed "*ranula pancreatica.*" Multiple small cysts, the result of obstruction of the smaller ducts, were named "*acne pancreatica*" by Klebs (1869). These two forms are uncommon and of no significance from the standpoint of diagnosis and surgical treatment.

Small cysts are recognized either at autopsy or in the course of upper abdominal operations for other conditions.

Large single or multi-locular cysts offer the best opportunity for diagnosis and treatment. They are said to be associated with obstruction of the large branches of the main duct and vary in size up to enormous proportions, cysts containing fifteen liters and more having been reported.

The walls of a retention cyst consist of dense fibrous tissue of

varying thickness. The inner surface may be lined more or less completely by a single layer of cylindrical epithelium, indicating the origin of the cyst from a dilated duct. The absence of this epithelial layer is no proof that it is not a true retention cyst as the action of the cyst contents may destroy the epithelium. Portions of pancreatic tissue may be found included in the walls of these cysts, indicating a true pancreatic origin. Continued increase in size of a cyst causes more or less destruction of pancreatic tissue even in those cases where the growth is mostly away from the pancreas. The outer surface of the larger cysts is traversed by greatly distended blood-vessels.

Proliferation Cysts.—These cysts are formed by proliferation of the duct epithelium and accumulation of fluid. They are of two kinds, benign and malignant. Benign cysts have much the same characteristics as multi-locular ovarian cystadenomas. They are multi-locular, with small cysts in the walls of the larger ones, and frequently there are papilliferous growths on the inner surface. As a rule such a cyst is lined with columnar epithelium, but this may have been destroyed by the cyst contents.

A few cases of malignant cyst have been reported. They occur as epitheliomas without regular form and are usually made up of numbers of small cysts.

Remnants of the Wolffian body are said to give rise to tumors resembling proliferation cysts of the pancreas. All of these growths are rare.

Traumatic Cysts.—Differentiation of these cysts depends more upon the etiological factor of injury than on any special pathological characteristics. Trauma may be a factor in causing either true or false cysts. The presence of blood in the cyst contents is not demonstrative of traumatism, since hemorrhage may occur as the result of rupture of vessels in the wall of a cyst already formed; on the other hand, a cyst undoubtedly due to injury may contain clear watery fluid with no macroscopic evidence of blood. Occasionally a cyst has old clots adherent to the wall, the result of previous hemorrhage.

The fact that traumatism will give rise to pancreatic cysts receives ample confirmation from both experimental and clinical observations. Lazarus (1904) produced a cyst by crushing the pancreas of a dog, forming a hematoma, which later became an encapsulated cyst containing 100 c.c. of watery fluid.

Traumatic cysts are very frequently situated in the lesser peritoneal cavity, having little or no direct association with the pancreas.

Hydatid Cysts.—Hydatid cysts of the pancreas are extremely rare and present practically no symptoms or physical signs by which they can be recognized definitely as hydatid cysts of the pancreas in contradistinction to other cysts of the pancreas or hydatid cysts of neighboring viscera.

Congenital cystic disease is rare and has little or no clinical significance; it is evidenced by multiple small cysts as in congenital cystic disease of other organs. The cases of cyst reported in children from six to fourteen months of age probably are not cases of congenital cystic disease, as they had all the characteristics of retention cysts.

Contents.—The appearance and character of the fluid contents of pancreatic cysts and pseudocysts vary considerably.

The contents may be almost any *color*, but usually are light brown, although in many reported cases the cysts have contained clear watery fluid. The presence of blood in varying quantities influences the color, according to the length of time that has elapsed since the blood escaped. The presence of enzymes also causes alteration in the color and may be responsible for the loss of the products of hemorrhage in the cyst contents. Enzymes may also be responsible for the presence of blood in the fluid by causing erosion of the vessels in the wall of the cyst.

The *character* of the contents varies from watery fluid to a thick syrupy or colloid substance too thick to run freely through an aspirating trochar. It is purulent if suppuration has occurred.

Macroscopic examination therefore offers very slight assistance in determining the origin of such a cyst. Opinions differ as to the value to be attached to the chemical analysis and microscopic examination.

Chemical Analysis.—The presence in the cyst contents of various ferments, rich in the usual digestive powers of the pancreas, is strong presumptive evidence that the cyst is of pancreatic origin. This is undoubtedly true if the fluid digests albumen, starch and fat. When all three ferments are present it is practically certain that the cyst communicates directly with the pancreas, but the presence of only one (unless it is the fatsplitting ferment) is not diagnostic. Many cysts of undoubted pancreatic origin do not contain all three ferments and each one of the three has been demonstrated in cysts of extra-pancreatic origin. The absence of pancreatic ferments in a true cyst is due to the fact that chronic disease of the pancreas interferes with its secretory function. Their presence in pseudocysts is due to communication with the parenchyma of the pancreas.

The other characteristics of pancreatic cyst fluids are: an alkaline reaction; a specific gravity from 1010 to 1020, although it may be

much higher; the constant presence of albumen; the frequent presence of cholesterin; the occasional presence of mucin; and rarely the presence of traces of urea.

Microscopic examination reveals red and white blood cells, epithelium, fat globules, necrotic tissue and frequently cholesterin crystals.

Symptoms and Physical Signs.—There are many cases in which the only recorded symptom is the appearance and gradual growth of an abdominal tumor, but these cases are exceptional. The presence of the tumor and the associated inflammatory and pressure changes usually result in marked digestive and constitutional changes. Pain also is a fairly constant symptom.

Pain.—This varies in severity from a feeling of discomfort or distention to attacks of severe lancinating pain common to the serious abdominal crises. Pain may be continuous, intermittent or continuous with acute exacerbations. At times the pain is severe enough to resemble acute intestinal obstruction, and often is accompanied by vomiting and collapse, which still further confuse the diagnosis. Very little dependence can be placed on the localization of the pain. Usually it is most severe in the upper abdomen, and is deeply seated. From this position it may radiate into either hypochondrium, to the back or the lower abdomen.

Pain may be present before there is any definite tumor or its appearance may be delayed until the cyst has reached a large size.

The relation between eating and the occurrence of pain is usually indefinite, but at times it occurs only after eating and may be associated with vomiting.

Vomiting depends on two factors, the severity of the pain and the interference with gastric function. It is almost always a concomitant symptom when pain is severe, and it has a close relationship to the occurrence of exacerbation. Vomiting is also a constant feature in those cases where the growth of the cyst directly compresses the stomach. It may be the delayed vomiting of obstruction of the pylorus, or reflex vomiting from gastric irritability.

Pressure Symptoms.—The grouping of these symptoms depends on the direction of growth of the cyst. Pressure on the stomach causes indigestion, flatulence, discomfort after eating, anorexia and finally vomiting.

Jaundice may be due to direct pressure on the common bile-duct or to associated pancreatitis or gall-stones. Constipation results from pressure on the colon and in a few recorded cases this has gone on to actual obstruction.

More remote results of pressure are ascites from obstruction of the portal vein, edema of the lower limbs from obstruction to the inferior vena cava, and hydronephrosis from pressure on the ureter.

Functional Disturbances.—The presence or absence of functional disturbances depends on the original cause of the cyst formation and on the degree of destruction of pancreatic parenchyma. If the cyst is caused by or associated with chronic pancreatitis the presence or absence of the signs of pancreatic indigestion depends on the extent of involvement. These signs, steatorrhea, azotorrhea, bulky pale stools, etc., are evidence of pancreatitis and not of pancreatic cyst, but their presence in conjunction with an upper abdominal tumor having the physical characters of a pancreatic cyst would be strongly corroborative evidence. Occasionally glycosuria is present.

The absence of signs indicating pancreatic insufficiency does not rule out a diagnosis of cyst of the pancreas, but simply means that there has not been sufficient destruction of glandular parenchyma to interfere with its function.

Loss of weight and strength is nearly always an accompaniment of large cysts and may be well marked even in those of moderate size. It results from various causes, notably from interference with pancreatic digestion, vomiting and gastro-intestinal disturbances and possibly from obscure metabolic changes.

Physical Signs.—The physical signs depend on the presence, direction and rapidity of growth of a cystic tumor originating in the upper abdomen between the ensiform and umbilicus. In the majority of instances the tumor occupies the middle line and projects to the left side more often than the right. Unless too small to reach the surface, the recognition of a rounded, smooth cystic tumor is easily demonstrated by abdominal palpation. The physical signs vary with the direction of growth from the point of origin.

Körte (1898) made *three classes of pancreatic cysts according to the direction of growth.* In addition there are cases that do not fit into any of these groups. The direction of growth is influenced by the origin of the cyst in relation to the reflections of peritoneum from the pancreas. Dilatation of the stomach and colon with air shows the relation of a cyst to these viscera. The degree of dilatation influences the amount of contact between the tumor and the abdominal wall.

1. *The first of Körte's groups* comprises those tumors that grow forward between the transverse colon and the stomach, displacing the latter upward and the former downward (Fig. 167). The amount

of displacement depends on the size of the tumor and the amount of air in the viscera. Cysts following this line of growth arise from the anterior surface of the head or body of the pancreas or are pseudocysts formed in the lesser peritoneal cavity.

2. *The second group* includes growths from the upper part of the anterior surface of the head or body of the pancreas which grow forward below the liver and above the lesser curvature of the stomach, displacing the latter downward (Fig. 168). This direction of growth is prone to occur in those patients subject to gastroptosis.

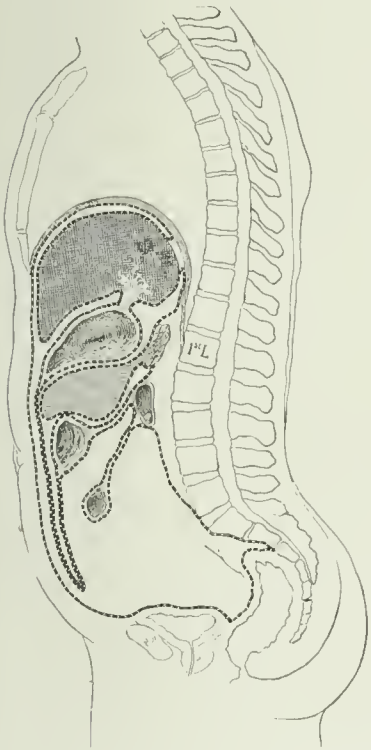


FIG. 167.—Cyst of Pancreas Presenting Beneath the Gastro-colic Omentum.

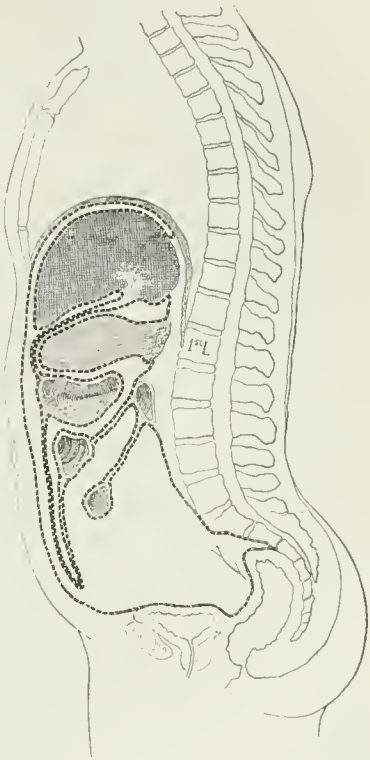


FIG. 168.—Cyst of Pancreas Presenting Beneath the Gastro-hepatic Omentum.

3. *The third group* comprises cysts from the tail of the pancreas. This lies on the left of the duodeno-jejunal junction and cysts developing here grow into the transverse mesocolon and displace the colon in one of three ways, upward, downward or directly forward. If the colon is displaced downward the tumor grows toward the left hypochdrium simulating a splenic growth. A cyst that comes out below the colon or pushes it directly forward, grows toward the midline

forming a tumor of the middle abdomen. Its relation to the colon is readily determined by distending the latter (Fig. 169).

The line of growth toward the *left hypochondrium* may also be taken by cysts arising from the body or tail of the pancreas above the reflection of the transverse mesocolon.

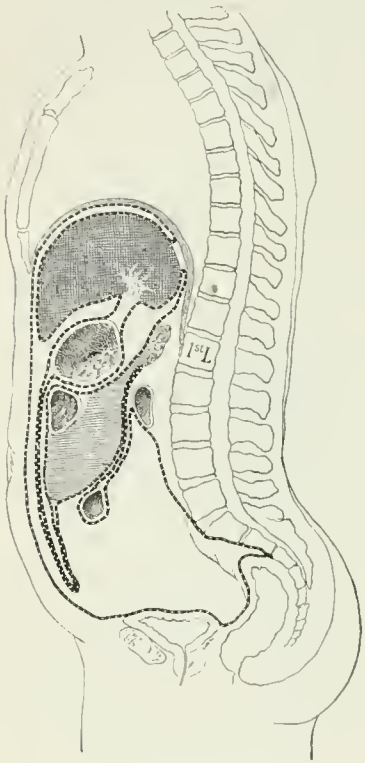


Fig. 169.—Cyst of Pancreas Growing into the Transverse Mesocolon.

Retroperitoneal growth into *either flank* may occur in cysts arising from the posterior surface of the pancreas. These cysts resemble tumors of the kidney or suprarenal.

Growth into the layers of the mesentery resembling a mesenteric cyst; growth foward from the head of the pancreas below the reflexion of the transverse mesocolon below the hepatic flexure of the colon, resembling a tumor of the cecum, ascending colon or right kidney; and growth through the foramen of Winslow into the general peritoneal cavity have all been reported. but the majority of cases fall into one or another of Körte's three main groups.

Mobility of an upper abdominal tumor does not exclude a pancreatic origin, and some cysts particularly those that arise from the tail of the pancreas, often are freely movable.

Pancreatic tumors as a rule *transmit the pulsation of the aorta*. This transmission ceases when the patient is in the knee-chest posture.

Disappearance of a cyst may be caused by rupture the result of an injury or exploratory puncture for diagnosis. Several cases have been reported in which the cyst disappeared as the result of discharging its contents into the bowel by way of the pancreatic ducts, or by means of a fistulous opening. This disappearance is followed by profuse diarrhea of material resembling cyst contents or saliva. These cysts may refill.

Diagnosis.—From the above description it is evident that there are no pathognomonic symptoms or physical signs of pancreatic cyst. A history of epigastric injury is often an aid in diagnosis. Even at

operation it is impossible to differentiate true from false cysts as their appearance and growth are so similar.

Diagnosis depends largely on physical signs and these may resemble those of growths of the liver, kidney, suprarenal, spleen, mesentery or ovary.

As a rule, the *history*, the *relation existing between the stomach and colon*, possibly the accompanying symptoms and signs of *pancreatic insufficiency*, make the diagnosis fairly certain in the majority of cases; but in those exhibiting eccentric forms of growth, particularly if unaccompanied by any symptoms referable to the pancreas, the diagnosis always is more or less uncertain.

Differential Diagnosis. *Mesenteric and Omental Cysts.*—As cysts of the pancreas may occupy either of these positions and are also at times freely movable, it is obviously impossible to differentiate certain cases.

Kidney and Suprarenal Cysts.—The resemblance of these growths to retroperitoneal pancreatic cysts is sometimes very close; and if urinary findings, ureteral catheterization, renal symptoms, X-rays and examination of the feces give no clue the diagnosis must be doubtful. Tympany behind the axillary line should indicate a pancreatic rather than a renal growth.

Cysts of the Liver.—Almost without exception these are echinococcus cysts, but they may closely resemble in physical signs pancreatic cysts growing forward between the liver and stomach. Inflation of the stomach might aid in diagnosis as it is much more likely to obscure a pancreatic than a liver tumor. Obtaining fluid by puncture should ensure a correct diagnosis, but this is attended with considerable danger. Exploratory operation is much safer and more certain.

Ovarian Cysts.—It should be the rarest possible occurrence to confuse ovarian and pancreatic cysts after careful bimanual examination under anesthesia if necessary. The presence of a normal uterus, tubes and ovaries in one, and in the other the recognition of a pedicle, with possible displacement of the uterus by traction or pressure and the difference in history make confusion of the two conditions very unlikely.

Enlarged Gall-bladder.—Close contact with the abdominal wall, respiratory movement if non-adherent, dullness continuous with the liver dullness, direction of growth, and the history serve to differentiate a distended gall-bladder from a cyst of the head of the pancreas even if the latter is accompanied by jaundice.

Cysts of the Spleen.—The history, direction of growth, physical signs, particularly the relation to the stomach, colon, left rib margin and area of normal splenic dullness serve to make confusion of these two very unlikely.

Prognosis.—Pancreatic cysts may persist for years without causing any symptoms except enlargement of the abdomen. These cases, however, are exceptional.

As a rule the course is progressive, symptoms becoming more pronounced with increase in the size of the tumor. Pressure symptoms become marked, interference with pancreatic function causes loss of weight and strength, and later probably diabetes.

Spontaneous or traumatic rupture may occur at any time and cause death by shock or peritonitis. Suppuration is not very infrequent. W. W. Ashhurst (1894) successfully evacuated two litres of pus by pancreatotomy.

Sudden enlargement accompanied by symptoms of shock indicates hemorrhage into the cyst. Of 160 operations for pancreatic cyst mentioned by Robson and Cammidge, recovery ensued in 140 cases, though eight of these patients died within a few weeks or months. Of thirteen patients under their own care eleven recovered after operation. Among eleven operations for pancreatic cyst by the senior author, there was one death, a mortality of 9 per cent. Four other patients have been under his care, in whom the diagnosis of pancreatic cyst was not confirmed by operation or autopsy: two of these patients went home somewhat improved in health, and two died before operation was permitted.

Treatment.—Medical treatment has no effect in staying the progress of the disease. Aspiration is contraindicated because of the danger of peritonitis or perforation of one of the large vessels in the wall of the cyst.

Complete extirpation is only occasionally possible, usually in those cases where the cyst arises from the tail of the pancreas or where it grows forward between the layers of the mesocolon. Excision is more difficult and has a higher mortality than incision and drainage and is nearly always impossible because of adhesions (Richardson). It was successful in the case of the patient whose history is detailed below (Fig. 170).

Incision and drainage (marsupialization) is the most suitable operation in the majority of cases. The incision is made over the most superficial portion of the growth and a rubber tube inserted into the cyst cavity after evacuation of the contents. The tube is

held in place by catgut sutures and leakage guarded against by a purse-string catgut suture tied after inverting the drainage wound and tube.

Occasionally drainage through the loin is easier than through the abdomen.

The cyst wall should be fastened to the abdominal wall but not to the skin.

FIG. 170.—Cyst of Pancreas Removed Entire with Pedicle and Some Adjoining Healthy Pancreatic Tissue. From a Patient in the Lankenau Hospital.

The presence of pancreatic juice in the drainage fluid indicates a direct connection between the pancreas and the cyst cavity. Care must be taken to avoid excoriation of the skin by the pancreatic juice in the discharge. Ointments with a mineral rather than an animal base are recommended since the latter is digested by the discharge. As a rule granulation tissue gradually obliterates the cavity and it closes completely, but occasionally a small fistula may persist for years. Antidiabetic diet, as recommended by Wohlgemuth, hastens the closure of the fistula (page 658).

Cyst of Pancreas; Excision. Recovery

J. M., aged 17 years. Admitted to German Hospital Dec. 6, 1912.

Chief Complaint.—Pain, sharp and constant, in epigastrium; anorexia, constipation.

Previous Medical History.—Negative.

Family History.—Mother and father living and well. Four brothers and five sisters living and well. Two brothers died in infancy.

Social History.—Eats and sleeps poorly. Bowels irregular. Denies venereal infection. Does not smoke or drink.

Present Illness.—For past seven years has had a gnawing, constant pain in stomach just to left of midline, 4.5 centimetres below the ensiform cartilage, and radiating to the back. It was made worse by eating—the greatest pain coming on one-half to one hour after eating his meals. One year ago the pain became more severe and he is now unable to work. He cannot sleep or eat anything but soft and liquid diet. November 16, 1910, he was operated on in another hospital for acute perforative appendicitis; he made due recovery, but his stomach pain persisted in spite of it. He has never vomited food or blood. Never has passed blood by his bowels, but has been constipated. His pain is present always, feels as if he had a boil on the coating of his stomach. He has no distention, no rigidity and peristalsis is fair.

Physical Examination.—Anemic Italian boy in great pain. Eyes and ears negative. Lips pallid. Tongue, clear, fissured. Teeth, good. Chest, fair development and expansion. Heart and lungs, surgically negative. Abdomen, muscular, seems wasted. No distention or rigidity. Peristalsis good. Sharp, gnawing, constant pain increasing one-half to one hour after eating, present 4.5 centimeters below the ensiform cartilage to the left of midline. It is aggravated on light palpation. It seems evident that there is an ulcer present. No pain in right iliac fossa. No palpable masses. No herniæ. No adenitis. Genitalia and extremities, negative. Blood pressure—systolic 110, diastolic 80.

Operation.—Dec. 9, 1912. Dr. Deaver. Upper right rectus incision. Peritoneum opened. Adhesions found between the great omentum and parietal peritoneum. Liver adherent to the parietal peritoneum. Peritoneum clamped to towels. Gall-bladder found normal and connected by adhesions to the hepatic flexure of the colon and the gastro-colic omentum. Adhesions also found between the omentum and the duodenum. Pancreatic lymph-nodes were enlarged. Pylorus patulous. Adhesions between the jejunum and the under surface of the transverse colon. Lesser peritoneal cavity opened through the gastro-colic omentum and mass found in the tail of the pancreas. Mass found to be a cyst the size of a small lemon in the tail. The cyst excised (Fig. 170). Rubber dam drainage. Opening in the transverse meso-colon and the gastro-colic omentum closed with single iodin gut. One piece of rubber dam to the stump of the pancreas. Wound closed to drainage.

Recovery was uneventful, and patient was discharged January 18, 1913.

CHAPTER XXIII

SURGERY OF THE SPLEEN

Anatomy

Position.—The spleen occupies a position in the posterior portion of the left upper abdomen behind and slightly to the left of the cardiac end of the stomach. Its long axis is nearly parallel to the course of the ribs. Its posterior boundary is at a point 3 to 5 cm. external to the vertebræ, and it extends to the mid-axillary line anteriorly. Its upper end is opposite the spine of the ninth dorsal vertebra, and its lower end is at the level of the first or second lumbar vertebra. The phrenic surface lies beneath the ninth, tenth and eleventh ribs.

Surface Anatomy.—The normal area of splenic dullness extends in the mid-axillary line longitudinally from the ninth to the eleventh rib, and transversely from the mid-axillary to the posterior axillary line.

Appearance, Size and Shape.—The spleen is of a dark red or purplish color and has an average weight of about 195 grams. The size varies somewhat, being greater at the height of digestion as a result of congestion. The shape depends largely on the surrounding viscera and is best ascertained by hardening *in situ*, by which means one inconstant and three constant surfaces are presented for study. These surfaces are named from the organs with which they are in contact.

The *phrenic surface* is the outer and posterior convex surface lying beneath the diaphragm. It ends in front at the anterior border, which is particularly well marked below the level of the hilum and contains one, two or three notches, a diagnostic sign in enlarged spleen. The anterior border separates the phrenic and gastric surfaces.

The *renal surface* rests against the anterior portion of the upper end of the left kidney and suprarenal capsule. This surface does not extend as high as the phrenic surface. The tail of the pancreas is sometimes in contact with this portion of the spleen, and sometimes with the gastric surface. The renal and phrenic surfaces are separated by the rounded posterior border of the spleen.

The *gastric* or *anterior surface* is concave and is in contact with the fundus of the stomach. This surface contains the hilum, a fissure

for the vessels, and at its lower portion is in contact with the splenic flexure of the colon, unless there is a basal surface.

The *basal surface* when present is a small flattened area on the inferior pole of the spleen resting on and being supported by the splenic flexure of the colon.

Blood-vessels.—The *splenic artery* is a branch of the celiac axis. It is relatively very large for the organ it supplies, and its course is quite tortuous. At the level of the tail of the pancreas it runs foward in the lieno-renal ligament, to break up into several branches which enter the hilum one above another and anterior to the veins. These vessels ramify through the connective-tissue trabeculæ of the spleen and do not anastomose. The branch that enters near the upper pole of the spleen is given off first from the main trunk and supplies several small branches to the stomach (vasa brevia). The gastro-epiploica sinistra is given off from the splenic artery just before it divides into its terminal branches.

The *splenic veins* are formed in the fibrous trabeculæ. They join to form several large branches which emerge from the hilum and unite to form the splenic vein behind and below the artery. The splenic and superior mesenteric veins unite to form the portal vein. This fact accounts for the splenic congestion and enlargement associated with cirrhosis of the liver.

The *lymphatics* emerge from the hilum and empty into nodes at the tail of the pancreas.

The *nerves* come from the solar plexus and enter the spleen with the arteries.

Ligaments.—The spleen is enveloped in peritoneum except where the ligaments meet at the hilum to form the pedicle. These ligaments are folds of peritoneum which transmit the blood-vessels (Fig. 196, p. 801).

When the surgeon's hand is introduced through an abdominal incision and passes over the anterior surface of the stomach to the fundus, it encounters the spleen. The fold of peritoneum thus felt, which joins the spleen to the stomach, is the *gastro-splenic ligament.* In it run the *vasa brevia*, branches of the splenic artery to the fundus of the stomach. If the surgeon carries his hand still further to the patient's left, it will pass between the spleen and the inferior concave surface of the diaphragm. The fold of peritoneum that arrests the fingers as they pass behind the spleen toward the spinal column, is the *lieno-renal ligament.* If one hand is placed in this situation, and the tips of the other fingers are placed anteriorly on the gastro-splenic ligament, the pedicle of the spleen, with all its contained vessels will lie between the two hands. If

the fingers are passed up over the external convex surface of the spleen to its upper pole they will here encounter the suspensory ligament of the spleen, the *lieno-phrenic ligament*, which binds it to the diaphram. If this is severed, the spleen may be drawn down from beneath the diaphragm and sometimes may be brought into the abdominal incision.

Relations.—From the description given above it is seen that the spleen is in relation with the following structures: *Externally* and above with the diaphragm, and above this with the pleura, the lung, and the ninth, tenth and eleventh ribs. *Anteriorly* with the stomach. *Internally* with the left kidney and suprarenal capsule. *Inferiorly* with the splenic flexure of the colon.

It is important to remember these relations to surrounding organs when dealing with an injury of the spleen since, especially in cases of penetrating wounds, one or more of the surrounding structures may be injured.

Anatomical Anomalies.—*Accessory spleens* are of two kinds, those consisting of true splenic tissue, and those having the structure of hemolymph glands. The former are true accessory or supernumerary spleens; usually they are joined by connective tissue to the anterior border of the spleen from which they have been cut off during fetal life. Occasionally these masses of splenic tissue are found free in the great omentum, in the transverse mesocolon or in the gastro-splenic omentum near the hilum of the spleen. Their significance is unknown. Accessory spleens resembling hemolymph glands have the same distribution as the true accessory spleens and in addition are said sometimes to be found within the tail of the pancreas. Usually they are about the size of a pea, and vary in number from fifteen to twenty, although much larger numbers have been reported.

Congenital absence of the spleen, is excessively rare, but has occurred.

Variations in Size.—The spleen may be very small, no larger than a walnut, and all variations from this to the normal size occur.

Lobulated and *abnormal shape* has been noted occasionally, but appear to have no surgical interest.

Movable spleen is a pathological condition and is described at page 728.

Physiology

Various functions have been attributed to the spleen, but our knowledge of none of them is very definite. Some theories are supported by a certain amount of experimental evidence while others owe their acceptance to the fact that they have not yet been disproved.

The functions of the spleen are best studied by considering first the results of its removal.

The results of splenectomy clearly demonstrate that the functions of the spleen, whatever they may be, readily are assumed by other organs. The changes following excision of a normal spleen can be studied only by animal experimentation, although a certain amount of useful information is obtained after splenectomy in human beings for injury to the normal organ. In the latter cases, however, the organism has to overcome the effects of severe injury and hemorrhage, as well as the loss of the spleen, and the various factors are hard to differentiate. Noguchi (1912) recorded the removal of a normal spleen because of its intimate adhesions to a lipoma which was being excised. The patient was a man forty-two years of age, and his blood did not return to normal for five or six years after the splenectomy; at first there was a decrease of the polynuclear leukocytes, and their place was taken by lymphocytes and eosinophile cells. The results of splenectomy for disease of the spleen give no reliable information, as the splenic function practically has disappeared and compensation has occurred before operation.

The *blood changes* following excision of the spleen may be summarized as follows:

Diminution of the red blood cells.

Disproportionate decrease in hemoglobin.

Leukocytosis.

Eosinophilia and lymphocytosis.

These changes are transitory, reaching their maximum in a few weeks or months; a gradual return to normal then occurs. But lymphocytosis and eosinophilia may develop late and persist for some time or may be absent. No conclusions of any practical value can be drawn from these changes. Infection is responsible for many of the abnormal results of splenectomy in human beings.

After splenectomy there is also developed an *increased resistance of the red-blood cells to hemolysis;* and *a lessened tendency to hemoglobinuria and jaundice* after adiministration of hemolytic agents (Bottazzi, 1894; Pearce, Krumbhaar and Frazier, 1918).

The Blood-forming Function of the Spleen.—The fetal spleen manufactures *red blood cells* but there is no direct evidence to show that this function is normally maintained in post-uterine life.

In cases of *severe anemia* there are in the spleen collections of cells resembling myeloid tissue, a fact which strongly suggests that, in these conditions, it is actively engaged in the formation of red blood cells

(Meyer and Heineke). The administration of pyridin to rabbits results in the formation of areas in the spleen which closely resemble the collections of hematogenetic cells in the normal rabbit embryo. Pyridin causes anemia by great destruction of blood and its mode of action probably resembles that of the cause of primary pernicious anemia (Morris, 1907).

In a case of *splenomegaly with sclerosis of the bone marrow*, reported by Donhauser (1908), the spleen contained islands of what he considered active hematoplastic tissue, the bone marrow having lost its bone-forming function as a result of sclerosis.

These observations are sufficient evidence to warrant the assumption that in certain pathological conditions the spleen reverts to its fetal blood-forming function. That the *regeneration of blood after hemorrhage* does not depend on the spleen to any extent is shown by the fact that regeneration takes place as quickly in splenectomized as in normal animals (Freytag, 1908).

While the red-cell-forming function of the spleen is disputed, there is little reason to doubt that this organ is concerned in the formation of *lymphocytes*, in common with the other lymphatic structures of the body.

Hemolysis and Blood Cleansing.—There is no direct evidence to prove that the spleen is a hemolytic organ, though this is generally believed. It contains large phagocytes in which are disintegrating red cells or particles of pigment, as well as pigment free in the pulp. This pigment deposit and red blood cell destruction are greatly increased in certain severe anemias. There is also free in the spleen a large percentage of organic iron. These facts suggest that the spleen either destroys the erythrocytes, or, more probably, acts as a mechanical filter and takes up the remains of the red blood cells, causes their disintegration and again puts the iron into solution.

Metabolic Functions of the Spleen.—*Organ of Iron Metabolism.* Experiments show that the total elimination of iron in splenectomized but otherwise normal dogs is considerably greater than in dogs with spleens. This relation is maintained whether the dogs are starved or well fed on meat. From these facts it may be concluded that the spleen is an organ of iron metabolism, serving to preserve for the use of the organism the iron which is liberated by the metabolism of the body.

Influence on Growth.—Grossenbacher concluded from the results of experiments on puppies, that the spleen exercises no appreciable influence on growth, differing therefore from the thyroid and thymus

in that its presence or absence does not definitely influence the course of life.

Protection of the Organism against Infection.—Hubbard (1909), after reporting his own experiments on the resistance of guinea-pigs to staphylococcus pyogenes aureus infection after splenectomy, and reviewing five other communications, concludes "that the removal of the spleen does not alter, practically, the individual's susceptibility to infection and that its functions in this respect, if they do actually exist, on its removal are readily taken up by other organs."

Almost nothing is known of the functions of the spleen and the cause of its enlargement in various infectious diseases and intoxications. This enlargement is supposed to indicate some protective action but definite knowledge is lacking.

Manufacture of an Internal Secretion.—There is a theory, supported by the experimental work of Schiff, Herzen, and others, that the spleen elaborates an enzyme which acts on the trypsinogen contained in the pancreas and converts it into trypsin. That this function, if it does exist, is of little moment is demonstrated by the results of experiments with the fresh pancreatic juice of splenectomized animals. It was shown that this contained trypsin in an active form (Pawlow).

In common with various other organs, the liver, pancreas, lungs, etc., the spleen contains a ferment; this is known as adenase, and is capable of converting adenin into hypoxanthin. Its significance is unknown.

Uric acid is present in the spleen and it has been suggested that it is formed as a result of proteid metabolism, but Chittenden and Mendel as the result of their experiments say, "there is no evidence that the spleen exerts any special influence on either carbohydrate or proteid metabolism in general."

Movements of the Spleen.—The nerves supplying the spleen are derived from the sympathetic; their stimulation causes contraction; and their section causes expansion of the spleen.

Roy (1880–82) showed that the spleen of the dog contracts and relaxes rhythmically about once a minute. It is supposed that these contractions keep up the circulation and make it independent of the general blood pressure. After a meal the spleen slowly expands, reaching its maximum size in about five hours, and then slowly returns to normal. Relaxation of the muscles in the trabeculæ and vasodilatation are supposed to cause this enlargement.

The relation between these movements and the function of the spleen has not been discovered.

General Consideration of Enlargements of Spleen

Most of the conditions which are of surgical interest cause an enlargement of the spleen; and it is convenient to consider the physical signs of such enlargements in this place, before discussing the different diseases of this organ.

As the spleen enlarges it emerges from beneath the ribs about the level of the ninth costo-chondral junction. Further enlargement follows the same line, and an hypertrophied spleen always is in the left abdomen until it reaches the level of the umbilicus. Further enlargement carries the splenic tumor across the middle line as well as downward on the left side toward the pelvic brim. The physical signs vary with the degree of enlargement.

Inspection.—If the abdominal wall is thin the enlarged spleen, particularly its lower border, may be seen moving with respiration below the costal margin. Otherwise inspection reveals only undue prominence of the left hypochondrium over the region occupied by the tumor. The enlargement may be so enormous that the whole abdomen is distended, but careful inspection usually shows this to be more marked on the left side. If the lower pole reaches the pelvis the tumor does not move with respiration.

Palpation.—The characteristic features of an enlarged spleen are its close apposition to the abdominal wall, a sharp inner border, and most important of all, interruption of its inner border by one, two or three notches. These are all readily felt if the organ comes out from under the rib margin to any extent. Splenic tumors always grow *forward;* they never produce fullness in the loin. Unless the spleen is anchored by peritoneal adhesions, it moves freely with respiration. Another very characteristic feature is continuance of the tumor up under the rib margin, closely applied to the abdominal wall. It is possible to insinuate one's hand between the ribs and the upper border of all but the very largest abdominal tumors, but splenic tumors unless prolapsed are too closely applied to the abdominal wall to permit this. Occasionally when there is marked perisplenitis a friction rub may be felt as the spleen moves with respiration, and adhesions may obscure the notches and twist the spleen so as to obliterate the sharp anterior margin. With the exception of these notches, an enlarged spleen usually is smooth and firm, although in certain infectious diseases it is sometimes too soft to be felt. The enlargement may be so slight that the edge is just to be felt with deep inspiration.

Percussion.—An enlarged spleen is dull to percussion up to the seventh or sixth rib or even higher in the mid-axillary line. The colon is displaced first downward, and later lies behind the enlarged spleen, so that any resonance due to it will be in the flank or loin.

Auscultation.—In the majority of splenic tumors auscultation is negative. If there is any perisplenitis there may be audible a to-and-fro friction rub. Very occasionally there is an audible murmur, hemic in origin, due to venous dilatation. In obscure cases it is well to outline the enlarged spleen by auscultatory percussion.

Differential Diagnosis.—Splenic enlargements have to be differentiated from various kinds of abdominal tumors, those in and about the kidney causing the greatest difficulty.

Pancreatic Cysts.—For physical signs and diagnosis see page 709.

Kidney Tumors.—New growths of the kidney scarcely ever come into close contact with the anterior abdominal wall, and even when they do they also cause marked bugling of the loin.

Palpation.—Kidney tumors have a rounded contour with no sharp, notched anterior border. They cause bulging and increased resistance in the loin when they are large enough to simulate splenic enlargements. The range of motion is much less. The anterior surface slopes away from the abdominal wall as it approaches the rib margin and, except in the very largest tumors the hand can be insinuated between the costal margin and the upper portion of the tumor. An enlarged spleen is closely applied to the abdominal wall, at its upper pole, and the hand cannot be passed above it. A renal tumor seldom is so large that it crosses the median line, but this not unfrequently occurs in cases of splenic enlargement.

Percussion.—The descending colon overlies the anterior surface of the kidney and is pushed forward when the kidney enlarges. This causes an area of resonance over the abdominal surface of the tumor, while there is dullness in the loin. The reverse is true in splenic tumors. But very occasionally the growth of a renal tumor pushes the colon outward instead of forward and the area of resonance is in the flank as in splenic tumors; in such cases the surgeon must rely on the results of palpation and on tests of the renal functions, including catheterization of the ureters, etc.

Auscultation is negative.

Other symptoms and physical signs are of importance. The surgeon should not forget that tumors of the kidney are not always accompanied by characteristic renal pain, and that acute attacks of perisplenitis may cause paroxysmal pain much resembling renal colic. The most impor-

tant differential signs are obtained by cystoscopy, catheterization of the ureters pyelography and careful examination of the urine. In certain cases of splenic enlargement a differential blood count clears up the diagnosis.

Suprarenal growths as a rule have the same physical signs as kidney tumors, except that the colon is very often pushed downward instead of forward. Hematuria frequently is present as a result of infiltration of the kidney by the growth.

Perinephric Abscess.—Apart from the evidences of suppuration, the physical signs resemble those of enlarged kidney more closely than they do those of splenic enlargement. The urinary and cystoscopic findings depend on whether or not the kidney substance is involved.

Ovarian Tumors.—Several cases are on record where a displaced spleen has been mistaken for an ovarian tumor, the mistake being recognized only after the abdomen was opened. When the spleen is in the pelvis and the notch cannot be felt by vaginal or rectal examination it is readily seen how such confusion might occur. A diagnosis of prolapsed spleen can be made only by feeling the notch in the sharp anterior border. A large ovarian tumor could scarcely be mistaken for a tumor of the spleen enlarging from its normal position. The following characteristics of an ovarian tumor are sufficient to make the diagnosis: The upper border of an ovarian tumor seldom is in actual contact with the left costal margin unless it reaches also to the right costal margin. Ovarian tumors grow upward from the pelvis and the first and most prominent enlargement is in the lower abdomen. They do not move with respiration, and have no sharp border with one or more notches. They extend further across the middle line and cause more symmetrical enlargement of the abdomen. Vaginal examination as a rule shows the tumor in close association with a normal sized uterus, and frequently the pedicle of the cyst can be felt through the rectum. There usually is an area of resonance between the upper border of dullness over an ovarian tumor and the normal area of splenic dullness.

Growths of the Splenic Flexure.—Annular growths of the colon in the neighborhood of the splenic flexure usually are malignant and give rise to symptoms of intestinal obstruction before a palpable tumor develops. Occasionally, however, a diffuse tumor forms in the left upper abdomen before symptoms of obstruction occur, and such a tumor may have to be differentiated from an atypical enlargement of the spleen.

A tumor of the splenic flexure has not the definite shape of an enlarged spleen, with its sharp anterior border showing one or more notches,

nor has it the same close apposition to the abdominal wall throughout its extent. It is usually dull to superficial, but resonant to deep percussion. If fixed with adhesions it does not exhibit the same degree of mobility during respiration as does the spleen. If not fixed its position changes to a marked extent with changes in the patient's posture. Sooner or later such a tumor gives rise to symptoms of intestinal obstruction and metastasis, but the diagnosis should be made and operation undertaken before these occur. Roentgenological study will usually exclude splenomegaly.

Tuberculous Peritonitis.—In the fibrous form of tuberculous peritonitis tumors of various sizes and shapes are found in different parts of the abdomen. When such a tumor mass is formed in the left upper abdomen and particularly if it is adherent to the spleen it may closely simulate in general outline a splenic tumor. Attention to the following points serves to distinguish one from the other:

The range of movement in a tuberculous tumor usually is limited by adhesions to the abdominal wall. Although the anterior border may be well defined it seldom exhibits a notch similar to those of the spleen. There usually is an area of resonance between the tumor and the normal area of splenic dullness. Moderate ascites often is present in tuberculous peritonitis, but may also occur in some forms of splenomegaly. Careful search nearly always will reveal other tumors or indefinite areas of consolidation in different parts of the abdominal cavity. The tuberculin reaction is of great value; von Pirquet's skin test is sufficient in very young children, but in adults the hypodermic injection of old tuberculin gives more accurate results.

Malignant Peritonitis.—In this condition the physical signs and symptoms are very much like those of tuberculous peritonitis, except that it is not likely that the tuberculin reaction will be positive. In most cases other tumor masses are readily discoverable in various portions of the abdominal cavity.

Fecal Impaction.—Except that it may give rise to a tumor in the left hypochondrium there is very little resemblance between a fecal impaction and an enlarged spleen. The irregular, indefinite shape, symptoms of temporary obstruction alternating with diarrhea, pitting on pressure, movability and absence of the usual physical signs of splenic enlargement, serve to make the differentiation comparatively easy.

Sarcoma of the Stomach.—Primary sarcoma of the stomach is rare. When it occurs it is frequently situated at the fundus, infiltrates the whole stomach wall, and may cause a very large tumor in the upper

abdomen. As previously noted (page 282) it is accompanied by enlargement of the spleen in about 15 per cent. of cases.

Its distinguishing characteristics are: resonance on percussion over the tumor; changes of position during examination and when the patient changes his posture; a position further to the right than is usual with an enlarged spleen, that is, the right border may be beyond the middle line although the tumor does not extend below the umbilicus; definite gastric symptoms; and, while there is a sharp border, it is not notched as a rule. The course of such a case is rapidly progressive to a fatal termination.

Retroperitoneal tumors are not easily confounded with those of splenic origin. Retroperitoneal growths usually present within the circle formed by the large bowel, offering to percussion a dull area surrounded by intestinal tympany. Inflation of the colon and stomach, examination in the Trendelenburg and knee-chest positions, should be adopted in obscure cases.

Pleural Effusion, when massive, on the left side, may occasionally cause inversion of the diaphragm, with development of a tumor in the region of the spleen. "The tumor has a peculiar bulky feel, is tender to touch, does not move with respiration, has the shape neither of the spleen nor of the kidney, and appears to have a deep attachment. Its true nature is revealed after aspiration of the chest, when it will be found to disappear" (Riesman, 1920).

Causes of Enlargement of the Spleen

There are two groups of cases associated with splenomegaly. (1) Those in which the blood changes are distinctive. (2) Those in which the blood changes are not distinctive.

A complete blood examination is essential in every case of splenic enlargement. A positive diagnosis never can be made without it. By this means Group 1 is differentiated from Group 2 with comparatively little difficulty. With the exception of certain cases to be considered later (see page 752) operative interference is absolutely contraindicated in cases included in Group 1, and therefore they have no place in the consideration of splenomegaly viewed from a surgical standpoint.

Group 1. Cases of Splenic Enlargement with Distinctive Blood Changes.—This comprises most cases of malaria, the leukemias, pernicious anemia, splenomegalic polycythemia, typhoid fever, and kala azar.

Group 2. Cases of Splenic Enlargement without Distinctive Blood Changes.—This comprises three divisions: (*a*) cases in which splenectomy is advisable or necessary; (*b*) those in which it is occasionally indicated; and (*c*) those in which it is contraindicated.

The first division, in which *splenectomy is advisable or necessary*, includes:

1. Movable spleen.
2. Cysts.
3. Primary tumors.
4. Primary tuberculosis.
5. Abscess.
6. Banti's disease and its subvarieties.

The second division in which *splenectomy is occasionally indicated*, comprises some cases of:

7. Hemolytic Icterus (Hereditary and family forms of splenomegaly).
8. Syphilis.
9. Malaria.
10. Pernicious anemia.
11. Cirrhosis of the liver.

The third division is of surgical interest only from the standpoint of differential diagnosis, since *splenectomy is contraindicated.* It includes enlargements due to:

12. Congestion (active or passive) and inflammation.
13. Infarct and thrombosis.
14. Infectious fevers.
15. Hodgkin's disease.
16. Amyloid disease.
17. Rickets.
18. Perisplenitis.
19. Pseudoleukemia.
20. Obstruction of the inferior vena cava above the entrance of the hepatic veins by mediastinal tumors, pulmonary fibrosis, chronic heart disease, etc.

The diagnosis of some of these from surgical conditions is obvious. The others will be considered with the conditions which they simulate.

Movable Spleen

A movable spleen is also known as a *wandering*, a *floating*, or an *ectopic* spleen. The terms *prolapse* and *splenoptosis* are also applied. A *dislocated* spleen is one which is fixed in an abnormal position.

Etiology.—Prolapse of the spleen occurs most frequently in women; thirteen of fourteen cases noted in our first edition were in women. Elongation of the ligaments always is associated with any marked degree of splenic mobility. This lengthening may be congenital or acquired. Of the congenital condition we know nothing. Acquired lengthening sometimes is said to result from trauma, but it is probable that the injury leads to abdominal examination, and that the discovery is then made of a prolapse of the spleen which has existed without symptoms for some time. It is possible, however, that acute displacements may result from rupture of the ligaments. Cases of movable spleen which produce no symptoms usually are discovered incidentally during examination for other conditions. General enteroptosis may include descent of the spleen, without necessarily any increase in the length of the pedicle. Increased weight is a factor in etiology, although in the majority of cases of splenomegaly perisplenic adhesions and hypertrophy of the ligaments keep the organ in place. Thus the enlarged spleen of leukemia, splenic anemia, malaria, etc., usually does not prolapse. Yet of fourteen cases of splenectomy for wandering spleen mentioned in the first edition of this work, four were associated with malarial hypertrophy and three with idiopathic enlargement. Elongation of the pedicle to 25 cm. is reported and it often is long enough to permit the spleen to drop into either iliac fossa, or into the pelvis.

Pathology.—A movable spleen may vary in size from normal almost to any degree of enlargement. The pathological conditions in such a spleen are of two kinds, those the result of the displacement, and those occurring independently. Among the latter are morbid changes the result of fortuitous diseases such as malaria and leukemia. These changes are characteristic and well known and need not be considered in connection with the pathology of movable spleen. Those changes produced by displacement are caused by twisting of the pedicle and interference with the blood supply.

Acute torsion occurs suddenly and the twist is tight, causing necrosis of the spleen and thrombosis of the splenic vessels, unless operation is immediately undertaken.

Chronic Torsion.—When twisting occurs slowly, the twist is loose and causes only gradual interference with the blood supply resulting in enlargement from congestion of the spleen, hemorrhage into its substance and consequent increase of fibrous tissue. In late cases these changes may be succeeded by sclerosis and atrophy with consequent diminution in size.

Intermittent attacks of twisting have effects similar to those seen in cases of chronic torison. Such attacks in a wandering spleen usually cause perisplenitis and the formation of adhesions to the structures with which it comes into contact.

The degree of twisting in reported cases varies from one incomplete to four complete turns.

Symptoms.—Simple uncomplicated mobility causes little or no disturbance and the diagnosis is usually made incidentally during routine abdominal examination. Occasionally indefinite gastro-intestinal symptoms are associated with splenic mobility and may be considered the consequence of such displacement. Intermittent attacks of jaundice and functional disturbances in surrounding organs may result from the traction of an enlarged movable spleen. Digestive disturbances, constipation and even partial intestinal obstruction may result from dragging of the pedicle. But the only definite symptoms directly attributable to prolapse of the spleen are those arising from twists of the pedicle.

Acute Torsion.—The primary and most important symptom of this condition is severe paroxysmal pain and tenderness in the left hypochondrium. The pain may give rise to nausea and vomiting and a certain amount of shock, with increase in the pulse rate. If unrelieved the obstruction to the blood supply causes necrosis of the spleen and the development of localized peritonitis. Death from sepsis or the formation of a localized abscess may result.

Chronic Torsion.—The twist in the pedicle not being so tight as to cause complete obstruction of the vessels, the symptoms are less sudden and severe in onset. Torsion through less than three-fifths of a circumference seldom produces symptoms. Intermittent attacks of pain associated with local tenderness are the only symptoms of moment. These attacks may occur spontaneously or result from some sudden exertion or movement. Attacks of perisplenitis cause the same symptoms and are the cause of the formation of the adhesions that fix the spleen in abnormal positions (*dislocated spleen*). Rectal tenesmus is caused by impaction of a dislocated spleen in the pelvis. Attacks of perisplenitis or twist of the pedicle usually are accompanied by elevation of temperature and increased pulse rate.

Diagnosis.—In uncomplicated cases of movable spleen the organ is not enlarged to any extent and presents well-marked characteristics. It retains its shape, a sharp anterior border, and one or more distinct notches. The normal area of splenic dullness is absent. By putting the patient in the dorsal position, the spleen can be easily pushed

up under the left rib margin, where it will remain until the patient changes her position.

Twist of the pedicle causes increase in the size of the tumor, which becomes acutely tender. Rigidity of the abdominal muscles is pronounced but not general, being confined to the area covering the displaced spleen. If there are no adhesions the characteristics of the splenic outline are recognizable, although a diagnosis of movable kidney is frequently made. As a rule the displacement, the swelling and the formation of an inflammatory exudate obscure the physical signs and render diagnosis difficult. Cases are reported in which a dislocated spleen has simulated appendiceal abscess, twisted ovarian cyst, and pus tube. If the spleen is impacted in the pelvis, the correct diagnosis is impossible unless the notch can be felt through the vagina or rectum, and even then the infrequency of the condition will cause a doubt as to the probability of such a tumor being the spleen.

Differential Diagnosis.—In non-adherent cases the only confusion in diagnosis arises from the question of movable kidney, the great frequency of which condition and its similarity in symptoms and physical signs make a mistake pardonable. But careful examination for the splenic notch, attempting to palpate the kidney indepenently of the abdominal tumor, the absence of the urinary changes which frequently result from a kidney crisis and the different range of mobility in the two organs, are signs to be looked for and given due weight in arriving at a diagnosis. The splenic artery is said to be palpable in cases of movable spleen, but this is not true of the renal artery when the kidney is displaced. Forgetting the possibility of a movable spleen is the most frequent cause of mistake.

The diagnosis of an adherent misplaced spleen usually is impossible, since inflammatory exudate obscures the splenic outline, and the history and physical signs point to inflammation of the organ normally occupying the area affected. Thus a mass in the right iliac fossa, associated with pain, tenderness, rigidity, fever and leukocytosis is more likely to be an appendiceal abscess than anything else. Likewise, if the tumor is in the pelvis it simulates ovarian or tubo-ovarian disease.

Treatment.—For those cases of movable spleen discovered accidentally, no operative treatment need be undertaken. The patient does not suffer from the condition and no harm is likely to arise. For those cases complicated with twists, perisplenitis and adhesions operative treatment is indicated and consists in splenopexy or splenectomy, preferably the latter (see page 799). Of seventeen recently reported operations for wandering spleen mentioned in the first edition

of this work (fourteen splenectomies, two splenopexies, and one exploratory operation in which nothing was done when the tumor in the pelvis was found to be the spleen), all the patients made uneventful recoveries. Splenectomy sometimes is very difficult on account of the adhesions formed between the spleen and various abdominal organs. G. G. Ross did splenopexy in one patient with ptosis of the spleen, in the German Hospital, but symptoms persisted and splenectomy was eventually required and proved perfectly successful. In our first edition we tabulated 106 cases of splenectomy for wandering spleen, with 7 deaths, a mortality of 6.5 per cent. Among 37 other cases of splenectomy for twisted pedicle, there were 10 deaths, a mortality of 27 per cent.

Cysts of the Spleen

The two main varieties of cysts found in the spleen are the **parasitic** (chiefly echinococcus) and the **simple,** or non-parasitic. One case of **dermoid cyst** of the spleen was reported by Andral in 1829. The entire subject was reviewed by Fowler (1913) who analyzed 82 cases of all varieties.

Echinococcus Cysts.—Among native Americans echinococcus disease is almost unknown. In 1901 Lyon collected 241 cases of echinococcus disease reported in North America. The nationality of 149 was determined and among these there were only two Canadians and one American, the rest being foreigners (136), negroes (10) and undetermined (92). Of the 241 cases the spleen was the seat of disease in nine (3.7 per cent.).

Pathology.—Involvement of the spleen may be primary or by extension from the gastro-splenic omentum. Any portion of the organ may be affected and it may contain more than one cyst, but each individual cyst is unilocular. The size of the cysts varies; very often they are so small as to remain undetected until autopsy, at other times they reach a size which necessitates surgical interference. Small cysts may be destroyed spontaneously and the remains may become calcified. Large cysts may rupture or become infected. Rupture leads to serve toxemia and peritonitis, which may be fatal, or to the danger of dissemination of the disease by the escape of living parasites, if the more serious complications do not occur. Infection results in the formation of an abscess, presenting no special characteristics.

Macroscopically and microscopically the cyst presents the same appearance and causes the same tissue reaction in the spleen as in other organs.

Symptoms.—The symptoms are those of simple cyst. The "hydatid thrill" is usually absent, but may be simulated by other conditions.

Differential Diagnosis.—Exploratory puncture seldom is justifiable on account of the danger of toxemia, infection and the escape of living parasites into the peritoneal cavity. The finding of cyst elements in puncture fluid is pathognomonic but their absence does not exclude the possibility of echinococcus disease. The information obtained by exploratory puncture is not of sufficient value to warrant the risk incurred. A complement fixation test may also be used, as in hydatid disease elsewhere in the body.

Prognosis.—Cysts of a large size are always a source of danger. Rupture or infection may occur and are very serious complications.

Treatment.—Splenectomy is the ideal treatment but may be impossible on account of extensive adhesions. Under these conditions formolization, as described at p. 569, followed by resection and drainage should be undertaken. Successful results from marsupialization have been reported. Among 29 cases of splenectomy for hydatid cyst, tabulated in our first edition, there were 6 deaths, a mortality of 20.3 per cent.

Simple or Non-parasitic Cysts. Causes.—Trauma is the most frequent cause of non-parasitic cyst of the spleen and is the only etiologic factor that is accepted without question. It gives rise to a subcapsular hematoma which subsequently may become a serous cyst from deposition of the solid elements.

Blood Cysts.—In addition to trauma, other causes of blood cysts are hemangeiomata (p. 735), hemorrhage into serous or lymph-cysts, and hemorrhage into the spleen during the course of infectious fevers, typhoid, malaria, etc.

Serous Cysts.—Serous cysts usually result from hematomas, but occasionally their origin is undetermined and several theories have been advanced to account for their development. It is held by some that a cyst may result from degeneration of portions of the peritoneal endothelium misplaced beneath the splenic capsule during embryonic life. Another theory is that some of the splenic pulp is extruded through a rent in the capsule and that a cyst is developed in the space that is left.

Lymph-cysts.—Lymph-cysts are due to the degeneration of lymph-angeiomata or to the occlusion of lymph-vessels and the escape of lymph.

Retention Cysts do not occur as there are no tubular glands in the spleen.

Pathology.—*Blood cysts* contain blood and blood remnants. The color varies according to the age of the cyst and the degree of alteration in the blood. *Serous cysts* are characterized by clear fluid contents, of low specific gravity (1003 to 1010), containing no albumen. *Lymph-cysts* contain fluid having the characters of lymph; it is clear, straw colored, has a high specific gravity and contains a large amount of albumen. Cholesterin in varying amounts is contained in the fluid of every splenic cyst. When it is excessive the cysts are known as *cholesterin cysts.*

Non-parasitic cysts may be uni- or multilocular, and vary in size from those which are of no pathological significance to those containing 2 gallons of fluid. The cyst walls also vary from those of extreme thinness, to the thickened and calcified walls of old cysts. The cysts are lined with endothelium, but this is not always demonstrable if the walls have undergone degeneration. Any portion of the spleen may be involved at first, but as the cyst develops it very soon becomes subcapsular. Rapid increase usually but not always is due to hemorrhage into the cyst. Huntington (1907) reported a case where a lymph-cyst increased from the umbilicus to the pelvic brim in three weeks.

Adhesions may form around the spleen and make operation very difficult. Unlike hydatid cysts, rupture or infection scarcely ever occurs in cases of simple cyst.

Symptoms.—Pain is the most noticeable symptom. It is due to three main causes: the weight of the tumor, stretching of the capsule, or attacks of perisplenitis. Pain from the weight of the tumor varies in degree and may be only a sense of fullness and discomfort. Sudden stretching of the capsule and attacks of perisplenitis cause severe pain. Other symptoms result from dragging on and interference with the function of neighboring organs. Gastric, intestinal, urinary, respiratory and cardiac disturbances are thus occasioned. In some reported cases the patients have had chills, although the malarial parasite was not demonstrable.

The *physical signs* are enlargement of the left upper abdomen, a palpable, smooth tumor emerging from beneath the left costal margin, and fluctuation. Small cysts cause no symptoms or physical signs and require no treatment.

Diagnosis.—The diagnosis is based on the physical signs, and on the existence of a cystic tumor growing from and intimately associated with the spleen. Cysts of the spleen have been mistaken for pancreatic cyst, movable kidney, cystic kidney, hydronephrosis and ovarian cyst. These conditions have been sufficiently described.

Treatment.—Surgical treatment is indicated whenever the cyst grows rapidly or is of a size sufficient to cause distressing symptoms. Excision of the cyst with repair of the raw area, or splenectomy are the accepted methods of treatment at present. Marsupialization and incision and drainage should be considered obsolete. Successful results have followed all forms of treatment, but splenectomy has been performed oftener than any other operation. Among twenty-five operations reported in our first edition, there was only one death.

Tumors of the Spleen

These are rare. Types of sarcoma are the least unusual, but are more often secondary than primary. Fibroma, chondroma, osteoma, hemangeioma, and lymphangioma have also been observed.

The proper treatment of primary tumors is splenectomy.

The senior author has done one splenectomy for primary *sarcoma*, in a woman 38 years of age. The patient left the hospital in good health 6 weeks after the operation, but her subsequent history is not known. Giffin (1915) reported that two splenectomies had been done at the Mayo Clinic for lymphosarcoma: one patient remained well for several years but finally died with metastases nine years after operation, while the second patient died in five months with generalization of the disease. These cases, in addition to those tabulated in our first edition, give a total of 18 splenectomies for sarcoma, with 3 deaths (16 per cent.)

Dowd (1915) collected 13 cases of *cavernous angeioma* of the spleen; splenectomy was done in 6 instances, with 2 operative deaths, 3 operative recoveries, and 1 case in which the result was not recorded. In Dowd's own patient death occurred 2½ months after operation from metastases to the liver; and this occurrence was noted in two other patients who were not operated on. Many cases of hemangeioma are cystic and hence are recorded with cysts rather than with tumors of the spleen. The lesion probably is developmental in origin, as with the angeiomas in other parts of the body.

Tuberculosis of the Spleen

Usually this is a secondary involvement, though a few cases of apparently primary infection have been recorded. The spleen is not much enlarged. The formation of a cold abscess is common. Splenectomy is the only efficient treatment, but seldom can be done if the disease is so far advanced as to make the diagnosis reasonably certain before the abdomen is opened. In our first edition we mentioned 15 splenectomies for tuberculosis, with 3 deaths (20 per cent.).

Syphilis of the Spleen

Though the development of the clinical picture of splenic anemia in patients with syphilis, and the fact that the syphilis appeared to be the cause of their symptoms, had been recognized by Marchand (1903), Osler (1914), Giffin (1916), and others, it remained for Norris, Symmers and Shapiro (1917) to claim that all cases of splenic anemia were syphilitic in origin. Most authorities, however, have not been willing to accept their view; but while recognizing that in many cases of syphilis of the spleen the clinical picture of splenic anemia may occur, nevertheless teach that other conditions (some known and some unknown) may also produce the same clinical picture.

Syphilis, both in the congenital and the acquired form, frequently affects the spleen; producing a diffuse inflammatory overgrowth of connective tissue with splenomegaly. In the congenital cases there results a condition resembling von Jaksch's disease; and in the later stages of the acquired disease the splenomegaly may be independent of cirrhosis of the liver, thus resembling the first stage of Bânti's disease.

When constitutional treatment proves ineffective in relieving the accompanying anemia, the spleen may be removed, and in cases where this has been done the improvement in the patient's condition has been immediate and striking. In Hartwell's patient (1913) the hemoglobin rose in two weeks from 25 to 80 per cent.

Abscess of the Spleen

Abscess of the spleen, or **acute suppurative splenitis,** may occur in the course of pyemia from any cause. In malignant endocarditis, infective emboli lodging in the spleen give rise to abscesses, which are usually multiple and small. Infection of infarcts the result of thrombosis of branches of the splenic artery gives rise to abscesses which may be of sufficient size to occasion definite physical signs. Cases of abscess of the spleen that are amenable to surgical treatment usually have been preceded by some acute infectious fever. Malaria, typhoid, dysentery, influenza, dengue, and relapsing fever have been reported as predisposing causes. Typhoid fever and malaria are the most frequent infections preceding the development of splenic abscess. The infecting agent in general infections varies with the original cause but in those cases where the spleen is the only seat of disease the colon bacillus is the organism most often found, although staphylococci are occasionally observed, and it is probable that the typhoid bacillus alone is capable of causing suppuration. The majority of reports do not

include bacterial findings. Occasionally the spleen may be involved in an abscess caused by extension from a neighboring focus of infection, such as empyema, subphrenic abscess, perforated gastric ulcer, perinephric abscess, etc. Hematomas and cysts (simple or parasitic) may become infected with resulting abscess formation. Neugebauer (1909) has reported a case following appendicitis.

Pathology.—Pyemic abscesses are multiple and small and have no distinctive characteristics. Those the result of infection of an infarct usually are single and they always extend to the periphery. The majority of abscesses amenable to surgical treatment (this includes particularly those resulting from infectious diseases) are single and of large size. When they reach the surface of the spleen they excite perisplenitis resulting in the formation of adhesions to surrounding structures. The spleen always is enlarged, due partly to the presence of the abscess and partly to the inflammatory reaction it sets up. In deep-seated abscesses the enlargement is uniform, but when they extend to the periphery the swelling is localized, except in the very largest abscesses with destruction of the whole splenic pulp. The abscess cavity contains pus cells, broken down blood and splenic pulp. These sequestra of splenic tissue have been particularly studied by Küttner (1907) who found them noted in 43 (37 per cent.) of 116 cases of abscess of the spleen which he collected. The wall of the abscess is formed by necrotic splenic tissue surrounded by an area of inflammatory reaction. Occasionally the reaction is sufficient to isolate and completely wall off small abscesses so that the contents eventually become inspissated and the area of infection becomes quiescent. Infiltration of the abscess wall with lime salts may occur, and in some cases overgrowth of connective tissue with absorption of the abscess has resulted, leaving a small depressed scar to mark the site of the original lesion.

An infected hematoma or cyst has its own pathological anatomy, to which is added that peculiar to abscess formation.

Symptoms.—*Pain* in the left hypochondrium is the first and most constant symptom. It varies in character from a dull ache to severe paroxysms of extreme intensity. Fever, chills and sweats are fairly constant accompaniments, but their significance may be masked by the original disease, as when an abscess develops in the course of malaria. Occurring in pyemia, infection of the spleen is indicated by acute pain in the splenic region and aggravation of the symptoms of infection, with the occurrence of chills, fever and sweats. Quite often an abscess develops gradually, with obscure symptoms, and comparatively little pain and fever.

Perforation of a splenic abscess causes symptoms that vary with the site of the rupture. General peritonitis results from perforation into the peritoneal cavity, although this usually is prevented by the presence of adhesions. Vomiting of pus and blood follows perforation into the stomach, empyema perforation through the diaphragm, and the passage of blood and pus by rectum a perforation into the colon. *Subphrenic abscess* due to disease of the spleen usually is located in the lesser peritoneal cavity. Among forty-four cases of suppuration in the lesser peritoneal cavity, tabulated by Michel and Gross (1904), seven were due to disease of the spleen.

The general systemic disturbances resulting from splenic abscess are those of suppuration anywhere, and their severity varies in individual cases. Hectic fever, leukocytosis, secondary anemia, gastro-intestinal disturbances, anorexia, loss of weight, etc., occur; but as these may be present in any form of infection they have no localizing value.

Physical Signs.—The physical signs are those of enlargement of the spleen, with tenderness and rigidity more or less localized when the abscess approaches the surface. A circumscribed prominence may be seen and felt in moderate-sized abscesses, but when very large the abscess occupies so much of the substance of the spleen that surface localization does not occur. If the tenderness is not too great, fluctuation may be obtained in certain cases; in others the tumor is soft and doughy in consistence, with irregular indurated edges; and in still others no area of softening can be made out. A friction rub may be heard sometimes when perisplenitis is present.

An abscess which develops toward the deeper surfaces of the spleen will cause no demonstrable localizing physical signs, except enlargement of the spleen.

Diagnosis.—Pain over the spleen is by no means pathognomonic of abscess as it occurs also in perisplenitis, embolism, cysts, enlargements in some acute fevers, etc. Leukocytosis is not a constant factor, both the actual and differential count being normal in many cases. Definite fluctuation is infrequent. There are, therefore, always certain cases with such obscure histories, symptoms and physical signs that diagnosis before operation is difficult or impossible.

Exploratory puncture never should be undertaken because of the danger of peritonitis.

Frank cases of splenic abscess present fewer difficulties. Pain and tenderness over an enlarged spleen during or following some infectious fever, accompanied by chills, fever and sweats and poly-

morphonuclear leukocytosis, indicate the probable presence of suppurative splenitis. If, in addition, a localized fluctuating swelling develops the diagnosis is certain. In those cases in which neither a local softening nor diffuse fluctuation is demonstrable, rapid and progressive enlargement of the spleen is a valuable sign, and when taken in conjunction with a history indicating infection, is very characteristic.

Careful attention to the previous history, the symptoms and course of the illness, and the physical signs indicate the correct diagnosis in a majority of cases, in fact in all but those whose obscurity renders accurate diagnosis impossible. At the onset, the condition frequently is mistaken for a relapse of the original illness (malaria, typhoid fever, influenza, etc.).

Abscesses that do not cause palpable enlargement are of no surgical significance. Very large splenic abscesses have to be differentiated particularly from perinephric abscess, and from pyo- and hydronephrosis.

Prognosis.—In abscesses of embolic origin the prognosis is that of the original condition, as a rule very unfavorable. Those amenable to surgical treatment are not the result of pyemia and give a much more favorable outlook. A number of recoveries following operation are reported. Recovery has occasionally followed spontaneous rupture and discharge, but the abscess usually reforms if the patient survives the rupture. In single abscesses arising from local conditions the prognosis depends on early recognition and prompt operation. Under these circumstances a large percentage of recoveries may be expected.

Treatment.—Incision and drainage is the operation usually undertaken and the one most generally applicable. Under certain circumstances splenectomy may be preferable, as for instance, when the abscess recurs after drainage. As a rule perisplenic adhesions render splenectomy impossible.

Johnston in 1908 collected nine splenectomies for abscess with eight recoveries. Perkins, in 1907, wrote that splenectomy had been performed successfully ten times for splenic abscess. All reported cases to which we have reference since 1908 have been treated by incision and drainage. Balfour (1917) refers to 27 operations for splenic abscess with 4 deaths (15 per cent.).

Banti's Disease or Splenic Anemia

This group includes splenic pseudoleukemia, primary splenomegaly with anemia, splenic lymphadenoma, idiopathic or cryptogenic

splenomegaly, splenomegalic cirrhosis of the liver and primitive endothelioma of Gaucher, although many investigators class the latter disease as a new growth. With this exception it is generally considered that all the other conditions represent different names for, or various stages of the same disease, but a great deal of confusion still exists in regard to the classification of diseases characterized by splenomegaly and anemia. From a surgical standpoint the conditions here included as stages of or different names for Banti's disease have a strong reason to be grouped together, apart from their symptoms and pathology, as they are all, except splenomegalic cirrhosis, greatly improved if not permanently cured by splenectomy. Cirrhosis is the last stage of Banti's disease and when it appears the time has passed when splenectomy could have had any effect on the progress of the disease. The relation of syphilis to Banti's disease was mentioned at p. 736.

Banti's disease was first described by him in 1882, and is characterized by great chronicity and three definite clinical and pathological stages:

1. *Simple enlargement* of the spleen.
2. Enlargement with *secondary anemia.*
3. *Cirrhosis of the liver* with splenomegaly.

Etiology.—Our knowledge of the etiology of Banti's disease does not rest on a firm basis. Many theories have been advanced but no definite causative agent is known.

Banti's own belief is that the disease is due to an unknown infective agent setting up changes of a non-inflammatory character. This agent is carried by the blood to the spleen and causes degeneration of the splenic pulp and follicles and hyperplasia of the reticulum; the latter is the more conspicuous feature and results in the development of the fibro-adenomatous appearance so commonly seen; the morbific agent, according to Severino (1911), is then carried away from the spleen in the splenic vein, which at autopsy always is found sclerosed.[1] Two theories as to the origin of this toxin are maintained; according to one theory this toxin is supposed to be elaborated in the spleen, which is recognized as the seat of primary disease; the other theory is that the toxin is formed as the result of disordered metabolism elsewhere in the body. It is not improbable that toxins may be formed in both ways. Banti favors the second theory of the origin of the toxin. Osler suggested an autointoxication of gastro-intestinal origin as the starting-point of the disease. Rolleston

[1] Warthin's views on this subject are mentioned at p. 743.

believed that the spleen is the site of a chronic infective or toxic process which occasions the pathological changes observed there and which subsequently inhibits blood formation and causes secondary anemia.

Discussing splenic anemia, Sutherland and Burghard (1910) advanced the theory that this condition is a functional disturbance of the spleen and not necessarily an actual disease. They explain the origin of the signs and symptoms as follows: Normally the endothelial cells of the spleen ingest old red blood cells. Loss of vasomotor control of the splenic artery, due to disease of the visceral sympathetic ganglia (Barr), causes overfilling of the spleen, and this results in hyperplasia and increased functional activity. In consequence of this increased activity the endothelial cells destroy both diseased and healthy red blood cells (Harris and Herzog). The quick regeneration of erythrocytes and hemoglobin after splenectomy indicates hemolysis and not diminished blood formation as the cause of the anemia. The rapid rise in the number of red blood cells suggests that the blood-forming organs, which have been working at high pressure to overcome the excessive destruction caused by the spleen, are able to regenerate the blood very rapidly after the spleen has been extirpated.

The *bacteriology* of Banti's disease is negative. Animal experiments and careful study of operative and postmortem specimens have emphasized the fact that, if it is an infective disease, the causative agent is still unknown.

There seems to be no necessary connection between the *previous medical history* and the occurrence of Banti's disease; the relation of syphilis to Banti's disease has already been discussed (p. 736); but malaria, other infectious diseases and alcoholism have not been proved to have etiological significance. Nor is there any evidence to show a *family predisposition* for the disease except in the case of the primary endothelioma of Gaucher. Several observers of the latter disease have reported a family incidence with a tendency to appear in several members of the same generation.

It seems safest to conclude that Banti's disease may be due to a number of different factors; but it remains for the future to identify these more definitely before any etiological classification can be made of the diseases now grouped together for practical purposes under the same name (Banti's disease).

Age.—Cases are reported at ages from six to seventy-two years but the great majority occur between twenty and forty-five years. The

age of Banti's fifty patients ranged from twelve to fifty-five years, two occurring before the age of fifteen years, seventeen between fifteen and twenty-five years, fifteen between twenty-five and thirty-five years, eleven between thirty-five and forty-five years, and five between forty-five and fifty-five years. The age of Osler's fifteen patients ranged from twenty to fifty-eight years; that of West's twenty patients from nine to seventy-two years. Sutherland and Burghard report a splenectomy for splenic anemia in a child six years old. It is probable that many cases begin in childhood and on account of the chronic course of the disease the patient does not come under treatment for some years. The senior author has recently operated on a female child twenty-two months old, with recovery. The differential diagnosis of the splenomegalies of childhood offers unusual difficulties on account of their number, variety and obscure etiology.

Sex.—The first series of cases of Banti's disease published showed a great preponderance of males: thirteen of Osler's fifteen, nineteen of West's twenty-four, and seven of Lyon's eight cases. More recent reports do not sustain these figures; thirty-two of Banti's fifty patients were females, and of Simonds's collected forty-eight cases, twenty-five were females and twenty-three males. Torrance collected thirty-six splenectomies for Banti's disease; seventeen of the patients were females, fifteen were males. In four the sex was not mentioned. Practically, therefore, both sexes are about equally affected.

No *race* or *country* is particularly associated with the occurrence of the disease.

Pathology.—The first changes occur in the spleen, and these attain their permanent character before any other pathological alterations appear. Then secondary anemia develops and finally the changes in the liver and portal system complete the morbid anatomy of the disease.

Spleen.—The spleen enlarges steadily but retains its normal shape and appearance. The average weight is from 1500 to 1750 grams but enormous hypertrophy is reported at times. The largest spleen in Simonds's collected cases weighed 4500 grams, and in Bovaird's case (1900) of endothelioma the spleen weighed 6250 grams.

Very commonly there are a large number of perisplenic adhesions, and these may give great trouble at operation, sometimes rendering splenectomy impossible.

The capsule and fibrous tissue of the trabeculæ undergo considerable hypertrophy, but the most marked change is hyperplasia of the reticular fibres without any marked change in the cellular

elements. The Malpighian corpuscles are overgrown with connective tissue and atrophied, and in some places have completely disappeared. In addition the whole spleen shows more or less passive hyperemia. There is also in every case some proliferation of the endothelium of the sinuses. Very often normal areas of splenic tissue with functionally active Malpighian corpuscles remain scattered throughout the substance of the spleen.

The amount of endothelial proliferation varies and may be so great as to cause the characteristic changes known as *primitive endothelioma of the type of Gaucher* (1882). In these cases the spleen as a rule is uniformly enlarged, but in Stengel's case a nodular growth was present. Microscopic examination reveals hyperplasia of the endothelium occurring in large masses. In addition there are areas of necrosis and hemorrhage and overgrowth of the reticular connective tissue. The liver, lymph-nodes and bone marrow also show these endothelial accumulations. The characteristic large vesicular cells with small eccentric nuclei block the sinuses of the spleen and lymph nodes, or are crowded about the liver lobules. The appearance of the spleen is strongly suggestive of a new growth; but the long duration, the uniform distribution of the areas of endothelial proliferation and the cure of the condition by splenectomy are strong arguments against malignancy. Pathologically, primitive endothelioma is a distinct and definite form of splenomegaly but clinically it has the characteristics of splenic anemia. The disease begins before the thirteenth year of life; the child slowly develops a tendency to submucous and subcuticular hemorrhages; and *extreme* enlargement of the spleen occurs. Enlargement of the liver is secondary but may be of considerable proportions. There is a brownish yellow discoloration of the skin, and often a peculiar yellowish wedgeshaped thickening of the conjunctiva. According to Pearce, Krumbhaar and Frazier (1918) there are only 17 authentic cases on record. Splenectomy was adopted in 10 cases, with 3 deaths.

Splenic Vein.—There is always some chronic sclerosing endophlebitis of the splenic veins and in some cases it is excessive, with calcification and stenosis. The portal vein may also be affected. Warthin (1910) claimed that such changes were the primary lesion, resulting from any cause of inflammation, and that the changes in the spleen were merely secondary to the passive congestion thus induced. This was the view previously taken by Hill (1909) and others; but it is not generally accepted.

Liver.—There is no change in the liver until late in the second

stage of the disease. From this time on the changes cannot be distinguished from those of Laennec's atrophic cirrhosis.

Symptoms and Physical Signs.—Banti's disease runs an extremely chronic course of from five to twenty-five years.

First Stage.—This is characterized by its insidious onset and long duration. There is gradual progressive and painless enlargement of the spleen, which may be accompanied by a sense of weight and discomfort in the left hypochondrium. The increase in size of the spleen continues until the lower pole is on a level with or below the umbilicus, but when the pelvic brim is reached growth ceases. The splenic tumor in these cases is as big as any except the largest of the leukemic spleens. There is no connection between the size of the spleen and the occurrence or severity of the symptoms. Splenomegaly is present for three to five years or longer before the onset of the symptoms of anemia which mark the beginning of the second stage.

Second Stage.—The early symptoms of this stage are those of simple anemia, pallor, weakness, dyspnea and palpitation. The grade of the anemia has no relation to the severity of the disease. With the development of these symptoms the blood usually shows changes of chlorotic type—diminution of red blood cells and hemoglobin, with a low color index. The hemoglobin may be diminished to 50 or even 40 per cent. In addition there is also leukopenia with relative lymphocytosis; the total count may fall to 1000–1500 per cubic millimetre. Leukopenia is very characteristic of splenic anemia, but in common with diminution in the red blood cells and hemoglobin is not necessarily present in all cases. These changes may be very slight or even completely absent even when the symptoms of anemia are well developed. Later in the disease the blood picture may be profoundly modified by hemorrhages.

Following the appearance of anemia, changes in the amount and quality of the urine occur. Banti associates the onset of the second stage with the appearance of urinary changes. The amount of urine diminishes to 1000 c.c. or less in twenty-four hours, and it contains urobilin and albumen intermittently.

The liver begins to enlarge, until it is readily palpable three or four fingers' breadths below the costal margin in the right nipple line. It is smooth and painless. There is no ascites or jaundice.

Gastro-intestinal hemorrhages, particularly hematemesis, may occur before the onset of cirrhosis. Forty per cent. of the blood from the stomach reaches the portal system through the vasa brevia and splenic vein, and hematemesis is the mechanical result of congestion of

the splenic vein. Other hemorrhages, subcutaneous, from the nose and gums, hematuria and melena may also occur. Digestive disturbances and diarrhea occasionally are observed.

The skin and conjunctiva may develop a grayish-yellow color, but actual pigmentation or jaundice does not occur in this stage.

With the approach of the third stage the liver diminishes in size. The second stage lasts from eighteen months to several years.

Third Stage.—The onset of the third stage is characterized by the development of ascites. This does not necessarily mean the presence of cirrhosis of the liver, since ascites frequently is seen before cirrhosis is demonstrable, as is shown by several operations and autopsy reports. Anemia and enlargement of the spleen are the causative factors under these circumstances. The ascitic fluid has all the appearances of a transudate. It reforms rapidly after paracentesis.

The liver continues to diminish in size and well-marked cirrhosis develops. The gastro-intestinal hemorrhages are aggravated. The urine is still further diminished and contains urobilin and at times bilirubin. The skin develops a certain amount of pigmentation in a few cases and late in the disease actual jaundice may occur.

Diagnosis.—The diagnosis of Banti's disease or splenic anemia in the early stages, before the appearance of the anemia, is impossible. Whatever the subsequent developments, at this stage the case is one of "idiopathic" splenomegaly. Even after the appearance of anemia the differential diagnosis presents many difficulties. Fortunately the technique of splenectomy is so simple, and within recent years the indications have been so extended that a great many of the conditions that might be confused with splenic anemia are now subjected to the same treatment, splenectomy.

The diagnosis, however, often may be made with reasonable certainty. The splenic enlargement first must be distinguished from tumors of other organs. This subject was considered at page 723. Certain other diseases must then be excluded. The most important of these are discussed below.

As **cirrhosis of the liver** is the terminal stage of Banti's disease, it is possible, in the later stages of the latter affection, that doubt will arise as to the causation of the splenomegaly. If symptoms characteristic of hepatic cirrhosis, such as hematemesis, ascites, jaundice and decrease in the size of the liver, appear after the splenic enlargement has existed for some time, clinically the case is Banti's disease in the terminal stage; although it must be remembered that these symptoms do not neces-

sarily mean that hepatic cirrhosis exists, as they may precede its development. Enlargement of the spleen occurring at the same time or after the changes in the liver and the onset of symptoms, indicates that the true condition is cirrhosis of the liver rather than Banti's disease. The history and physical signs may enable one to determine the form of cirrhosis which is present.

That this degree of simplicity in reaching a diagnosis is not always observed, is shown by the confusion that still exists in the classification of these cases. The three forms of hepatic cirrhosis most likely to be mistaken for splenic anemia are the syphilitic, the alcoholic and the hypertrophic of Hanot.

In *Syphilitic Cirrhosis* of the liver the previous history, the Wassermann reaction, and the result of treatment are the main points on which reliance may be placed in reaching a diagnosis. Moreover, the enlargement of the liver due to syphilis is irregular. Leukopenia is absent and in some cases there is a persistent but moderate leukocytosis. It must be remembered that a positive Wassermann reaction is not necessarily diagnostic, as there is no reason to suppose that a man with Banti's disease might not have had syphilis previously. Nor is the size of the spleen a sign to be depended upon, as this varies from moderate to great enlargement just as in Banti's disease. The anemia of syphilitic cirrhosis may be very severe. Extreme anemia of undetermined origin always suggests syphilis.

In *Alcoholic Cirrhosis*, the small liver, big spleen and anemia suggest Banti's disease with recurring hemorrhages.

The age of onset, alcoholic history, early appearance of symptoms of cirrhosis and the late development of splenomegaly will serve for differentiation from all except the most rapidly developing cases of Banti's disease.

In the *Hypertrophic Cirrhosis of Hanot*, the great enlargement of the liver and the marked jaundice usually are sufficient to indicate that the enlargement of the spleen is secondary, even if the time of the occurrence of splenic enlargement in relation to hepatic enlargement is not known.

The question of *splenectomy in the cirrhoses of the liver* has already been discussed in connection with the latter subject (p. 557).

Hemachromatosis.—This is a rare condition of biliary and splenic enlargement associated with hemolysis of unknown origin. Pigmentation of the skin and internal organs results from the deposit of an iron containing pigment. Hepatic and splenic enlargement occur synchronously.

Pernicious Anemia.—Cases of pernicious anemia with great enlargement of the spleen are not very uncommon and in certain stages readily may be mistaken for splenic anemia. If the anemia is extreme, the color index high, and if nucleated red blood cells and poikilocytes are present, the case is much more likely to be one of pernicious anemia than of Banti's disease, even though the spleen is enlarged to the pelvic brim. Even if the changes in the blood characteristic of pernicious anemia are absent, the presence of extreme anemia is against Banti's disease unless there has been a recent severe hemorrhage to account for it. Even in the late stages of Banti's disease it is uncommon for the proportion of hemoglobin to fall below 40 per cent. unless there has been recently a severe hemorrhage. Morris Lewis reported (1908) a case of Banti's disease in which the hemoglobin was 90 per cent. a short time before the onset of hemorrhage and 18 per cent. a few days later. The history and blood picture usually serve to differentiate the two conditions, and confusion will be less apt to occur if it is borne in mind that a tremendously enlarged spleen does not necessarily rule out pernicious anemia.

Hodgkin's Disease.—A splenic form of Hodgkin's disease, characterized by anemia, enlarged spleen with lymph-adenomatous masses but without enlargement of the superficial lymph-nodes, has been described by some, but it is very questionable whether such a condition exists.

Malarial Hypertrophy.—Severe secondary anemia with enlarged spleen as a sequel of malaria presents many of the characteristics of splenic anemia. The previous history usually is definite enough to enable one to avoid a mistake in diagnosis. If repeated blood examination is negative, exploratory splenic puncture, as a rule, reveals the malarial parasite, but is open to the objection that it exposes the patient to the risk of severe hemorrhage. On account of the anemia and the danger of rupture from slight degrees of traumatism splenectomy is becoming increasingly popular as a treatment for "ague cake" spleen.

Leukemia is diagnosed by the blood picture. During remissions when the blood returns to normal, the diagnosis is difficult; but observation of the patient for a short time reveals the true condition.

Syphilis is not often a cause of symptoms and physical signs resembling splenic anemia. The Wassermann reaction and the result of treatment in doubtful cases are sufficient to establish the diagnosis. In adults this splenic enlargement usually but not always is secondary to syphilitic affections of the liver, while in children syphilis causes primary splenomegaly. D'Arcy Power (1908) reported a successful

case of splenectomy for simple hypertrophy, which turned out to be syphilitic in origin. Hartwell's successful case has already been mentioned (p. 736). Gumma of the spleen is of pathological rather than surgical interest.

Amyloid spleen always is secondary to causes, such as suppuration, tuberculosis, etc., which are easily demonstrable.

Tumors and **tuberculosis** of the spleen are discussed at page 735.

Splenomegaly in Childhood.—Except the hypertrophy occurring in *acute* infectious diseases, enlargement of the spleen in infancy and early childhood is a condition of obscure etiology. Congenital syphilis especially, and rickets, disturbances of metabolism and intestinal disorders of doubtful classification are all considered etiological factors in this group. It is very likely that some are cases of true splenic anemia; but on account of the frequency of splenomegaly in childhood from other causes, and the uncertainty of the blood picture, such a diagnosis always is doubtful.

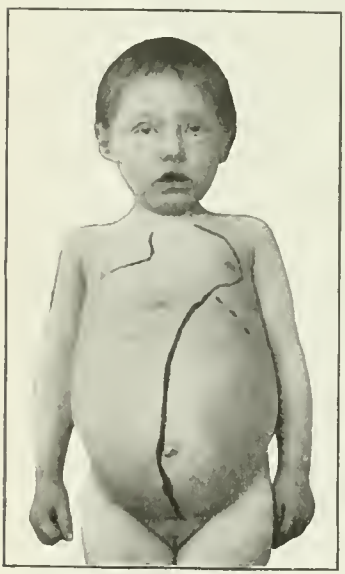


FIG. 171.—Splenomegaly of Undetermined Origin in a Child aged 3½ Years. (*Ashhurst.*)

Pseudoleukemia infantium of von Jaksch (1889), which usually appears in the second year of life, is characterized by marked splenomegaly and usually by moderate enlargement of the liver (Fig. 171). The red blood cells may be reduced to 1,000,000 and the hemoglobin falls disproportionately, giving a low color index. The blood shows normoblasts, poikilocytosis, leukocytosis (15–20,000), and relative increase in lymphocytes. Krumbhaar (1918) regards the disease not as an independent condition, but thinks it represents an atypical response of the infantile hemopoietic system to such diseases as leukemia, pernicious anemia, rachitis, syphilis, Banti's disease, hemolytic jaundice, etc. A history of congenital syphilis is present in about 50 per cent. of cases, and is regarded by Norris, Symmers and Shapiro (1917) as the cause of the disease. Stillman (1917) collected 6 cases of splenectomy with recovery and relief of symptoms, but in most cases medical treatment gives very good results, and even in severe cases iron, arsenic (arsphenamin) and mercury cause improvement and eventual cure in a certain pro-

portion of patients. One of the successful cases of splenectomy was in a child aged 15 months (Graff, 1908).

Prognosis and Treatment of Banti's Disease.—Medical treatment is absolutely worthless as regards the cure or arrest of the disease. With the most skillful administration of drugs or other therapeutic agents, such as the X-rays, the course of the disease is steadily toward a fatal termination. Splenectomy is the only form of treatment that offers any hope of permanent cure. As the operations and reported cases multiply and as the after-results are studied, it is becoming more and more evident that surgery effects an absolute cure in the first stage of the disease in every patient who makes an operative recovery. In the second stage the primary mortality is higher and the percentage of permanent cures is less. In the third stage the disease is too advanced to expect any good results from splenectomy, but Talma's operation may be undertaken at this time with moderate hope of success in removal of the symptoms of cirrhosis. One point should be thoroughly understood: ascites and slight or transient jaundice occurring in the course of Banti's disease do not absolutely contraindicate splenectomy, because they may occur before cirrhosis of the liver is present. The latter is the only contraindication to splenectomy, and it is not an absolute contraindication. Banti reported ten operations on his own patients and twenty collected operations with the following results:

Splenectomy for Banti's Disease	Banti's Cases		Collected Cases	
	Operations	Cures	Operations	Cures
First period	2	2	2	1
Second period	6	5	16	8
Third period	2	0	2	1

From January 1, 1908, to January 1, 1912 there were reported thirty-seven operations for Banti's Disease with nine operative deaths, a mortality of 24.3 per cent. Krumbhaar (1918) collected a total of 183 splenectomies for Banti's disease, with 28 deaths, a mortality of 15.4 per cent. Five cases in the hands of the senior author have all been successful. Mayo (1919) reports from the Mayo Clinic 61 splenectomies for Banti's diseases with 7 deaths, a mortality of 11.7 per cent.

Splenectomy is recognized as the only rational treatment for Banti's disease, and better results are to be expected in the future

with earlier resort to operation. Splenectomy should be undertaken as soon as the diagnosis is made, and the knowledge that the disease can be cured by operation should bring home to every physician the importance of early diagnosis.

The success of splenectomy seems to indicate that the spleen is closely associated with the causation of the changes characteristic of the second and third stages; but its real *modus operandi* is not understood. Mayo thinks its success depends chiefly on relieving the liver of an overload by cutting off all the blood normally delivered to the liver through the splenic vein: this is about one-third of the total portal circulation, and in cases of splenomegaly must be a considerably larger proportion. But is it not rather the *concentration* of poisons that is reduced by splenectomy? All the substances which entered the spleen through the splenic artery remain even after removal of the spleen, unless excreted elsewhere; but after splenectomy they no longer are delivered to the liver in concentrated form, but only through the hepatic artery and superior mesenteric vein after much dilution by their circuit through the body.

Splenocleisis.—Under this name Schiassi (1905) described an operation for Banti's disease in which the enlarged spleen is not removed, but is packed all around with iodoform gauze. This is to be allowed to remain for from six to ten days, by which time sufficient adhesions will have formed to ensure reduction in size of the spleen by their ultimate contraction. The large veins formed in this way are supposed to divert the blood from the spleen into the abdominal wall. Mugnai adopted this method in one case, but the patient died from internal hemorrhage the next day.

Hemolytic Icterus

Hemolytic jaundice occurs (1) as a *congenital, familial disease* and (2) as an *acquired disease of adult life.* The former was described in 1900 by Minkowski as "congenital acholuric icterus;" and is much commoner than the acquired form of the disease which is known by the names of Hayem (1898) and Widal (1907): of 159 cases studied by Krumbhaar (1918), 104 belonged to the familial type and only 55 to the acquired. Chauffard (1914) drew particular attention to congenital syphilis as a cause, though one of his patients acquired syphilis in adult life many years after his hemolytic jaundice first appeared;[1] in

[1] The occurrence of infantilism, dwarfing, etc., in some of these patients probably is an evidence of congenital syphilis.

other cases he incriminated tuberculosis, or at least claims that instances occur in those already tuberculous. He refers to those suffering with the congenital form of the disease as being more icteric than sick, and it is a fact that many such patients live for many years with very little inconvenience. Mayo (1919) suggests that as the spleen destroys excessive numbers of erythrocytes and thus inundates the liver with blood pigment, this renders the bile viscid, and predisposes to gallstone formation (60 per cent. of his patients with hemolytic icterus have had a complicating cholelithiasis); while gallstones, with the accompanying infectious cholangeitis, predispose to or may actually produce the biliary cirrhosis which often exists. It is on this account that the disease is so often confused with the so-called Hanot's biliary cirrhosis, a disease whose existence is doubted by Mayo (p. 554).

The fundamental symptoms are: (1) splenomegaly, (2) anemia, (3) jaundice. Patients come under treatment on account of the anemia; the large spleen may give no trouble and often passes unnoticed; while the jaundice, which rarely is more than an extreme sallowness of complexion, is "acholuric," and is unattended by the usual symptoms of obstructive ("choluric") jaundice (itching, slow pulse, clay colored stools, etc). Bile pigment is found in the blood, not in the urine except during exacerbations of the disease, known as "crises of deglobulization" (Widal, 1908), when urobilinuria may develop. During the worse phases the spleen grows larger, the urobilinuria increases, and the jaundice, if already present, deepens. During the better phases of the malady the red blood cells vary between three and four millions; in the bad phases they fall below one million. The color index usually is below 1; the hemoglobin may fall even lower then 20 per cent. The red blood cells show anisocytosis, poikilocytosis, and polychromatophilia. There usually are some cells with granules, and there may be even normoblasts. The resistance of the red blood cells is diminished: the initial hemolysis may be 0.60 and the total hemolysis 0.34. The white blood cells vary from 4500 to 11,500, usually being about normal. The differential count varies much from time to time. Myelocytes are seldom found, and only in the phases of intense anemia. True jaundice may not develop for many years; always is slight, but grows worse during the phases of severe anemia. The liver often is enlarged, especially during the phases of severe anemia.

In the congenital cases, or at least in childhood, splenectomy is not as definitely indicated as in adult life. In the latter class of

cases, according to Banti (1912), death occurs in from six to twelve years unless splenectomy is done, and this is the only measure which has any effect. Of 48 patients on whom splenectomy was done, only 2 died (Elliot and Kanavel, 1915): three patients were alive and well at periods of 27, 11, and 6 years respectively after operation; and 9 other patients, followed for 6 months after operation, remained in good health. Of 27 patients from the Mayo Clinic (1919) treated by splenectomy, only 1 died; all the others who were not in the terminal stages of the disease, were cured.

Malarial Splenomegaly

In these cases also, as in those of syphillis of the spleen, specific treatment occasionally fails to relieve the symptoms, and splenectomy may then prove beneficial, relieving the distressing symptoms due to the immensely enlarged spleen itself, and permitting seemingly more effective employment of antimalarial remedies. The statistics of splenectomy for malarial splenomegaly cited by Krumbhaar (1918) are twenty years or more old and though the mortality at that time was very high (20 to 25 per cent.) it does not appear that later operations have been more successful, though no extended series of cases appears to have been reported. In our first edition (1913) we tabulated 165 cases, with 41 deaths, a mortality of 24.8 per cent.

Pernicious Anemia

Krumbhaar (1918) writes: "The most important disease, from the point of view of its greater frequency and greater severity, to which splenectomy has been applied is pernicious anemia. The striking improvement that has been shown to follow removal of the spleen in such diseases as hemolytic jaundice and Banti's disease naturally led to an extension of this clinical procedure to allied conditions. In 1913 three investigators—Eppinger, Decastello, and Klemperer working independently, tried splenectomy as a therapeutic measure in pernicious anemia." Though they were induced to employ splenectomy for different reasons, they agreed in the expectation that removal of the spleen should favorably affect the course of this otherwise hopeless disease: Eppinger expected to secure a diminished output of urobilin and other evidences of hemolysis; Decastello seems to have regarded the improvement which followed splenectomy in somewhat similar conditions as sufficient to warrant its employment in pernicious anemia; while Klemperer was influenced by the clinical observation

that splenectomy in the healthy individual (for rupture of the spleen, etc.) was sometimes followed by polycythemia.

Krumbhaar, whose studies in this field no one can afford to ignore, has collected 153 cases of splenectomy for pernicious anemia in which the results are known: there were 30 deaths (19.6 per cent.) soon after operation;[1] 99 patients (64.6 per cent.) were improved by having their spleens removed, and only 24 (15.7 per cent.) showed no improvement. He found that at the end of six months after operation, of 53 patients no less than 44 continued to improve, 9 had already relapsed, but none who had survived splenectomy for more than six weeks had died in the interval. The followining table shows the results in cases traced by Krumbhaar for one and two year periods:

LATE RESULTS AFTER SPLENECTOMY FOR PERINCIOUS ANEMIA

	After one year	After two years
Number of patients traced	27	6
Still improved	11	3
Relapsed	7	2
Died subsequently	9	1
	54	12

As to the selection of cases for operation, we cannot but agree with Krumbhaar, who points out that though splenectomy has occasionally rescued seemingly moribund patients and restored them to comparative health for many months, yet that its effect is more certain and lasting, and the danger of the operation is less, if it is employed at as early a stage of the disease as possible. Especially favorable cases, he finds, are those patients with clinically enlarged spleens, icteroid appearance, and increased urobilin output, but without increased resistance of the erythrocytes. Splenectomy, on the other hand, he regards as distinctly contraindicated in cases where the bone-marrow is persistently aplastic; and it is not apt to prove beneficial when spinal cord symptoms have already developed.

Before splenectomy is undertaken in cases of pernicious anemia, it is desirable to await one of the periods of apparently spontaneous improvement which frequently occur; and to employ as adjuvant to this spontaneous improvement *transfusion of blood*. Splenectomy may then be undertaken with fair prospect of having the patient survive and securing such an improvement in his general health as to justify the risk.

[1]Mayo (1919) reports from the Mayo Clinic 50 splenectomies for pernicious anemia with 3 deaths (6 per cent.).

After splenectomy for pernicious anemia it is usual to have what is known as a "blood crisis"—the appearance in larger quantities in the blood of normoblasts, megaloblasts, reticulated erythrocytes, Jolly bodies, etc. Subsequently, as the blood returns nearer to normal, the hemoglobin and red blood cell count rise, but the color index usually remains high. In no case has a permanent cure of the disease been noted.

Myelocytic Leukemia

The mortality of splenectomy for this condition in 51 reported cases (1918) was 86 per cent. But after preliminary radium treatment of the enlarged spleen, it has been adopted at the Mayo Clinic in 20 cases, with only one death (Giffin, 1918); so it is apparent that this disease also is coming into the domain of surgery.

Cirrhosis of the Liver with Splenomegaly

The adoption of splenectomy in certain cases of cirrhosis of the liver attended by splenomegaly was discussed at p. 554.

Injuries of the Spleen

These may be divided into cases of *subcutaneous rupture*, almost always due to indirect violence, or to blunt force if the injury is direct, and *open wounds*, especially gunshot and stab wounds.

Disease of the spleen associated with enlargement frequently is a predisposing cause.

Subcutaneous Rupture.—If the spleen is normal a very great degree of trauma is necessary to rupture it. Being run over by vehicles, falls from a height, kicks and crushes are the usual causes of subcutaneous rupture. Violence may be applied directly to the spleen beneath the costal arch or indirectly through the ribs. In the latter case, fracture is not a necessary concomitant as the spleen often has been severely injured although the ribs remained intact. Rupture from indirect violence has also followed blows on the right side and even falls on the feet. If the spleen is enlarged it is easily ruptured by an insignificant degree of traumatism, such as tightening a belt, coughing, lifting, etc. This violence may be so slight that the case appears almost one of idiopathic rupture, but in the majority of cases a history of some slight injury is obtainable. There are, however, a certain number of cases in which rupture has occurred when the patient was lying quietly in bed. These are examples of true spontaneous rupture, although they are the result of advanced disease of the spleen, such as tuberculosis, or more particularly malaria.

In the tropics rupture of a malarial spleen is a common cause of sudden death. Labor or eclampsia may be the exciting cause of rupture if the spleen is enlarged.

Pathology.—Laceration of the splenic substance varies from a slight subcapsular rupture of the pulp to complete disorganization. The spleen may be divided into two or more pieces or it may be completely torn away from its vessels. Hemorrhage is the most important factor. If the capsule is torn the blood is poured out into the general peritoneal cavity and the spleen shows only the rent in its substance. If the capsule is untorn (subcapsular rupture) the blood collects beneath it and forms a hematoma. As the pressure increases, it may cause cessation of the bleeding or rupture of the capsule. In the former case a hemorrhagic cyst is formed, in the latter, a frank rupture. If clotting occurs and bleeding stops, the future condition of the spleen depends on the degree of injury. If any of the main vessels are involved necrosis will follow. If only the splenic pulp is torn, the wound may heal, or, as is more likely, softening of the clot will lead to secondary hemorrhage in a few days.

Site of Rupture.—If extensive injuries involving a large portion of the splenic substance are excluded, the site of rupture occurs five to six times more frequently on the internal surface than on all the rest of the surfaces collectively. Frequently the laceration extends from the inner to one or more of the other surfaces. If the rupture is limited to the internal surface immediately behind the hilum the bleeding may be confined to the lesser peritoneal cavity.

Rupture of Enlarged Spleen.—The same lesions occur as in the normal spleen, and the difference in morbid anatomy is that the spleen shows the changes characteristic of the pathological process to which it is subject.

Injury to Other Organs.—On account of its position, rupture of the spleen frequently is accompanied by injury to one or more of the surrounding organs, stomach, liver, intestine, kidney, pancreas and suprarenal gland. These injuries vary in severity as do those in the spleen. Fracture of the ribs and bruises on the surface of the body may indicate the point of application of the causative force, but very often there is no external evidence of injury.

Penetrating Wounds.—Stab wounds vary in depth and breadth according to the size of instrument and its depth of penetration. Gunshot wounds may cause great disorganization of the spleen or only small wounds of entrance and exit.

Injury to the surrounding structures is even more frequent than in subcutaneous rupture, particularly in gunshot wounds.

Thévenot (1910) collected eighty-one cases of gunshot and stab wounds of the spleen, and twenty-four of them were complicated by wounds of one or more of the surrounding viscera. The viscera injured were the

Stomach	in 14 instances.
Liver	in 5 instances.
Kidney	in 4 instances.
Intestine	in 5 instances.
Omentum	in 2 instances.
Lungs	in 2 instances.

In several cases more than one other organ was injured. In addition, the diaphragm and pleura are involved more frequently than any of the viscera. Among 54 cases of gunshot wounds of the spleen studied by Wallace (1917), only 32 were uncomplicated by other visceral injuries.

Symptoms.—As the symptoms from subcutaneous and penetrating wounds do not differ very much, they may be considered altogether.

They vary from sudden and immediate death, to the gradual or sudden onset of symptoms seventy-two hours or more after the injury. Sudden death may not be attributable solely to the splenic injury but may be due chiefly to complicating lesions. When the onset of symptoms is delayed, it is probable that the original injury produced a subcapsular hematoma which subsequently ruptured; or a primary complete but small rupture may have been sealed temporarily by clotting, and the late symptoms may be caused by dislodgment of this clot with secondary hemorrhage. In either case the symptoms, although appearing late, are the same as those that occur immediately after the injury.

Sudden, severe pain occurs in the left hypochondrium accompanied by a sense of tearing. The symptoms of internal hemorrhage appear with a varying degree of rapidity. Subsequently the pain in the left hyponchondrium varies in severity from a more or less continuous dull ache, with acute exacerbations, to pain so insignificant as to be almost negligible. Levy (1908) called attention to the value of sharp pain in the left shoulder region as a sign of rupture of the spleen. Breathing and moving aggravate whatever pain is present. The time of appearance of the signs and symptoms of internal hemorrhage depends on the rapidity of the bleeding; very often a large amount of blood escapes slowly into the abdomen before appreciable symptoms appear. The two most important signs are increasing pallor and rise in the pulse rate with diminution of its volume. Subsequently the patient exhibits

a rapid, running pulse, restlessness, vomiting, air hunger, and other signs of severe internal hemorrhage. Abdominal distention with rigidity more or less localized to the left upper quadrant may be present, and with these signs there may be increase in the area of splenic dullness and dullness in the left flank instead of the normal resonance. Dullness in the flank when present often is not a shifting dullness. Frequently all these signs are absent although there is an extensive laceration. Tenderness over the spleen usually is demonstrable either from in front or behind.

Rupture in the course of typhoid fever as a rule is mistaken for intestinal perforation. The rupture is manifested by pain beginning in the splenic region, by marked abdominal rigidity, followed by distention, with small rapid pulse, and extreme toxemia. A drop of the temperature to subnormal may occur at the time of rupture, as in cases of intestinal perforation, but, as also in these cases, the temperature rises as peritonitis develops. Bryan (1909) collected 35 cases of spontaneous rupture of the typhoid spleen.

Rupture of a malarial spleen causes the rapid appearance of shock from internal hemorrhage, and in most cases this is quickly fatal.

Diagnosis.—History of an injury, pain in the left hypochondrium, symptoms of internal hemorrhage, dullness in the flank and localized rigidity, when present all together leave very little doubt of the diagnosis. Injury to the kidney is ruled out by the absence of blood in the urine. Intestinal injury gives rise to earlier and more marked abdominal rigidity, which usually is more generalized; and the symptoms of internal hemorrhage are likely to be less severe unless the mesenteric vessels are torn across. The site of the blow usually excludes liver injury; but in certain cases, and particularly those where the splenic rupture is due to indirect violence, differentiation is practically impossible. Rupture of the pancreas rarely occurs without injury to the surrounding organs and it frequently is involved with the spleen. The stomach and gall-bladder rarely are injured except in the case of penetrating wounds. Stomach injury may lead to hematemesis; otherwise the symptoms are those of intestinal rupture. Laceration of the gall-bladder resembles injury to the liver except that the symptoms of hemorrhage are not marked.

As the treatment for all these conditions is the same, laparotomy, time ought not to be wasted in attempting to differentiate, but operation should be undertaken as soon as it is evident that there has been visceral injury. If the surgeon waits for shock to subside few patients will be saved from death.

Prognosis and Treatment.—The treatment of splenic injuries is immediate laparotomy. This is followed by packing of the wound, by its suture, or by splenectomy, according to the extent of the injury to the spleen. The mortality of cases in which no operation is done is 95 per cent. or more. The operative mortality is about 33 per cent.; this, however, does not represent the full mortality of rupture of the spleen, as many patients die too soon to allow time for operation. There are several reported recoveries from injuries that could not be distinguished clinically from rupture, but there must always be a large element of doubt in the diagnosis when the patient recovers without operation.

Operative Treatment.—The incision should be made in the epigastrium parallel to and just to the left of the middle line, so as to permit thorough exploration and treatment of concomitant injuries.

The operation selected depends upon the position and extent of the injury. Splenectomy is more often undertaken than all other operations combined, but repair of the wound, plugging with gauze, and suture of the omentum to the wound have all been recommended and performed with success.

Splenectomy is done most frequently because it is the most certain way to stop the bleeding; because the operation is easily performed in the majority of cases; and, more particularly, because the greater number of splenic injuries are of such a nature that they cannot be treated in any other manner with a reasonable hope of success. There were reported in the first edition of this work 227 cases of splenectomy for injury with 155 recoveries and 72 deaths, a mortality of 31 per cent.; and more recent reports indicate that the death rate continues at least as high; but it must be remembered that in a large proportion of the fatal cases there were other visceral injuries. According to Wallace (1917) in 32 uncomplicated cases the mortality was 50 per cent.; and 63 per cent. in 23 complicated cases.

Suture.—Even when the injury is small it is very difficult to control the hemorrhage with sutures. In a report of 134 cases of rupture of the spleen, Lotsch (1908) gave the following results: splenectomy, 118 cases with fifty-two deaths; splenorrhaphy, eight cases with three deaths; tamponade, eight cases with two deaths. Kirschner (1909) described a method of encapsulating the wound by sewing the omentum to the edges. He operated on four cases with one death, and in this case operation was refused until the patient was almost moribund.

Tamponade.—The advantage of plugging with gauze is that the spleen is preserved. This undoubtedly furnishes a strong argument

in its favor, but the control of hemorrhage is uncertain in all but the most favorable cases. Tamponade is particularly indicated in those cases where the presence of perisplenic adhesions renders splenectomy difficult or impossible. It may be advisable to soak the gauze in adrenalin solution.

Temporary Clamping of Vessels.—Sheldon (1910), as a result of experimental work on dogs, advises the use of rubber-covered clamps applied to the pedicle for four hours. They should then be loosened and if hemorrhage does not recur may be removed. They may be reapplied for another two hours if hemorrhage persists. In his experiments hemorrhage did not recur when the clamp was removed in four hours. The splenic wound is not treated. By this means the vessels empty themselves without clotting. If the wound is packed the clot extends back through the vessels to the clamp and necrosis might result.

Ligation of the splenic Artery through the Gastro-hepatic Omentum, was suggested by John Gerster (1915) as an easy method of controlling hemorrhage from the spleen, and it might be of value in some traumatic lesions where exposure of the splenic pedicle proved difficult. (Fig. 197.)

Aneurysm of the Splenic Artery

Splenectomy for Aneurysm of the Splenic Artery was done successfully by Winckler (1905), who refers to one similar operation by Selten, and says that Ponfick had found an aneurysm of the splenic artery four times at autopsy. Villard and Murard (1912) attempted splenectomy in one case, but adhesions rendered this impossible. The pedicle of the aneurysmal tumor had to be clamped to check bleeding, and the clamps were left in place, as ligation was impossible. The patient died in ten days from secondary hemorrhage. In both Winckler's and Villard's patients the chief symptoms were recurring attacks of pain of some years' duration. In both the operation was exploratory, a correct diagnosis not having been made before opening the abdomen.

CHAPTER XXIV

TECHNIQUE OF OPERATIONS

General Considerations.—In the First Part of this work (p. 309) we have covered so fully the subject of *preparation of the patient* and *after-treatment* that nothing additional need be said.

Operations on the Gall-bladder and Bile-ducts

Position of the Patient.—The patient is placed in the dorsal position with a sand bag or firm pillow beneath the back at the level of the liver. This pillow should be about 45 cm. long, and cylindrical in shape; its diameter should be from 10 to 15 cm. Instead of a pillow a special operating table may be used, provided with an adjust-

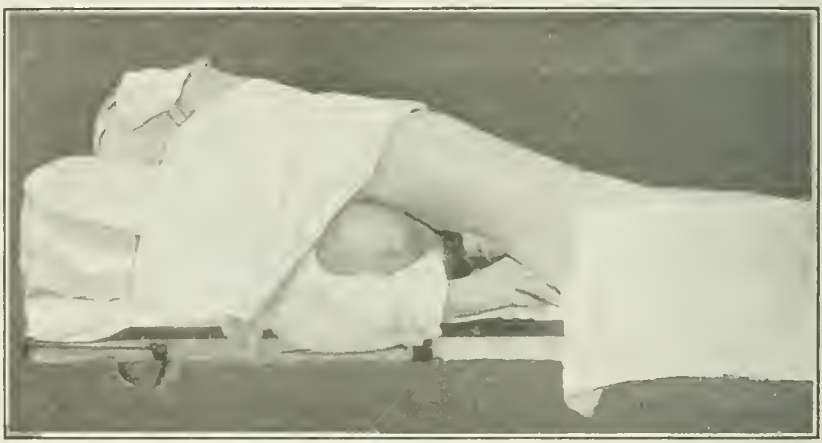


Fig. 172.—Patient in Position for Bile-duct Operations.

able "bridge." When properly placed this pillow raises the lower dorsal and upper lumbar spine away from the table in such a manner that the liver and biliary passages are pushed forward, the costal angle is widened, and the intestines fall by gravity toward the pelvis. A pillow or two should be placed beneath the head and shoulders to relieve tension on the neck and to raise the head to a proper level for the administration of the anesthetic (Fig. 172). Elliot, of Boston, who introduced this position in 1895, also raised the head of the table (reversed Trendelenburg position); and in difficult cases this has much to commend it.

Incision.—Numerous incisions have been advocated as best adapted for exposing the gall-bladder and common duct. Some of these are objectionable, because disregarding important structures in the abdominal wall, especially the nerves, and thus predisposing to hernia or muscular atrophy. Particular attention was called to this danger from nerve injury by Assmy in 1899 though it had been pointed out by Kocher before 1894.

Courvoisier's Incision (1890) runs parallel to the costal margin from the ensiform to the flank; all structures of the abdominal wall are divided in the same line. Though this incision gives ample exposure it is objectionable because it cuts all the lower intercostal nerves. *Kocher's incision* (1894) is similar to Courvoisier's but a little shorter and more transverse.

Lawson Tait (1879) and *Riedel* (1892) used a longitudinal incision through the right rectus muscle; and this is sufficient for ordinary cases of cholecystostomy.

Langenbuch's incision (1896) consists of a longitudinal cut along the right semilunar line, when necessary combined with an oblique incision along the costal margin to the ensiform; or a longitudinal incision through the rectus (as in Riedel's method), combined when necessary with an oblique incision along the costal margin inward to the ensiform or outward as far as necessary.

Mayo Robson's incision (1897) is similar to Langenbuch's second method—a longitudinal incision through the outer third of the right rectus muscle, with an upward and inward extension along the costal margin to the ensiform (Fig. 173). This is the incision used habitually by the senior author, and he finds it adequate even in difficult cases. Yet Terrier, than whom no more skillful and experienced operator on the biliary tract has ever lived, declared (1905) this incision not sufficient, and for difficult cases preferred Kehr's incision.

Kehr's incision (1905), known as the *Wellenschnitt*, and the *Bayonet incision*, begins at the ensiform, passes down 3 to 4 centimetres in the mid-line,then cuts the right rectus as far as its outer third (parallel to the costal margin) between its linea transversa and the umbilicus, and thence continues longitudinally downward as far as the umbilicus or lower. There is no doubt that this immense incision gives very excellent exposure of the deeper biliary passages, but we have never felt inclined to adopt it, having found we could accomplish everything necessary through a simpler incision. If Kehr's incision is carefully sutured, and no wound infection occurs, it does not appear to predispose the patient to hernia more than the other incisions employed.

Gosset (1912) found that among 142 operations in which drainage had been employed after the use of Kehr's Wellenschnitt, a hernia resulted only in four instances. It cuts no more nerves than Robson's incision, and divides the rectus in precisely the same manner, only a little lower. Of late Kehr has abandoned the lower limb of his original incision, so that the method he now adopts somewhat resembles that of Czerny (p. 763).

FIG. 173.—Scar Resulting from Robson's Incision. (*Ashhurst.*)

Collins's incision (1908) "begins at the inner edge of the right rectus muscle 1 or 2 inches (2.5 to 5 cm.) from the ensiform cartilage, and extends diagonally downward and outward to the outer edge of the right rectus close to the level of the umbilicus. It cuts through the skin, fat and anterior wall of the sheath of the rectus. A short transverse incision about 1 inch in length may be made inward from the upper end of the oblique incision through the skin, fat and linea alba; and a similar one through the linea semilunaris at the lower end. The rectus muscle is then separated from its sheath. When the muscle is thoroughly freed from its sheath, except at its outer border, it is easily retracted outward and allows the posterior sheath and the peritoneum to be incised in the same direction as the skin and anterior wall." Collins points out that transverse division of both linea alba and linea semilunaris permits the wound to gape widely,

but that suture is easy and repair very efficient. The junior author habitually employs this incision, being influenced by the fact that it does not damage the motor nerves to the rectus, and being near the midline gives adequate exposure of the ducts. Terrier's maxim, that biliary surgery tends to become more and more *canaliculaire*, should be kept in mind.

Czerny's Incision.—Czerny described his *Winkelschnitt* in 1892; and Kocher finally (1911) adopted a modification of it as his own "normal incision for difficult cases as did Perthes (1912). The original incision ran for from 5 to 7 centimeters in the mid-line, above the umbilicus, and bent just below the umbilicus to the right, cutting the right rectus muscle transversely. As employed by Kocher, Perthes, and other surgeons in recent years, the longitudinal part of the incision has been longer, extending from the ensiform, close to the midline, almost to the umbilicus, where it cuts the right rectus transversely.

Sprengel's Transverse Incision.—Sprengel (1910) described a transverse incision for operations on the bile passages, which has been adopted by Gosset (1912) in France, and for which Moschcowitz (1916) has made himself the protagonist in this country.

Sprengel's transverse incision divides the right rectus muscle directly across at whatever level seems desirable, depending upon the distance that the liver extends beyond the costal margin. If the suspensory ligament of the liver is in the way, it should be divided. Kehr objected to the use of this incision as consuming too much time. He speaks of fifteen or twenty minutes as required before the abdomen is opened. Gosset found it took him only from two to three minutes to make the whole incision. Only in exceptional cases is it necessary to divide part of the left rectus, to gain sufficient exposure. Division of both semilunar line and linea alba permits wide retraction. It is well to follow the advice of Perthes (1912), to suture the anterior sheath to the rectus muscle on both sides of the incision before the muscle is divided: this prevents both retraction of the transversely cut muscles and annoying bleeding. This incision gives ample exposure, there is little tendency for prolapse of the intestines even when the patient strains; and probably the chief advantage of all, it is a wound easily closed, inasmuch as there is no tendency for it to gape during contractions of the oblique muscles. Moreover, the posterior sheath of the rectus holds sutures better after transverse than after longitudinal division.

Bevan's incision (1899) begins near the ensiform cartilage, runs

parallel with the costal margin as far as the linea semilunaris, then extends along this line about 10 to 12 cm. and finally is carried transversely outward, thus making an elongated S-shaped incision. In simple cases neither the inner nor outer extension of the longitudinal incision need be employed. It is then the same as Langenbuch's original incision. When both the inward and outward extensions are used, it does more damage to the abdominal nerves than any incision, except Courvoisier's, ever devised.

As stated already, of all these incisions the senior author prefers Mayo Robson's. In simple cases the longitudinal portion alone is used, splitting the fibres of the rectus muscle parallel to their course. If more room is needed, the incision is continued inward and upward, parallel but not too close to the costal margin, into the space between the ensiform process and the ribs. If the incision passes too close to the ribs difficulty will be experienced in suturing it, on account of the retraction of the flat muscles on the outer side of the wound. This incision of Robson's cuts only a few of the upper abdominal nerves, and only after they have entered the rectus muscle.

When the time comes to suture any of these upper abdominal incisions, closure is much facilitated by removing the sand pillow or support from beneath the patient, and thus relaxing the abdominal wall. When this is done, it should not be forgotten that the bile passages will recede at once from the anterior abdominal wall, and that the drainage may become displaced if not sufficiently long or firmly fixed.

After the abdomen has been opened, recognition of the anatomical landmarks and relative position of the viscera is of the greatest importance As a preliminary to exploration, the upper right abdominal region should be *isolated with pads of gauze*, wrung out of hot saline solution: the first is passed toward the median line pressing the duodenum and stomach away from the liver and gall-bladder; a second depresses the colon; while a third is passed into the subhepatic space, to the right of the incision. Even after the diseased area has been walled off in this way, recognition of the anatomical landmarks of the biliary tract is often the most difficult step encountered throughout the entire procedure, especially in those cases where repeated attacks of inflammation have bound the various structures into an almost homogeneous mass by dense adhesions. These adhesions must be separated sufficiently to allow proper surgical treatment of the lesions, but we do not advocate unnecessary interference with adhesions which often are nature's barrier against infection of the general peritoneal cavity.

The adhesions should be approached from the convex surface of the liver, the border of the liver in the region of the normal site of the gall-bladder being freed first. From this point of entrance to the anatomy of the biliary passages, careful separation of the adhesions should be continued until the symptom-producing lesions have been uncovered.

When possible, the lower border of the liver should be drawn downward and then lifted upward into the abdominal incision slightly rotating the organ around an antero-posterior axis, so as to turn its

FIG. 174.—Rotation of the Liver to Expose the Gall-bladder and Bile-ducts.

inferior surface toward the left of the patient (Fig. 174). This can be done in most cases where the anterior border of the liver is not held too firmly in place by adhesions. It may be facilitated by division of the falciform ligament, as recommended by Judd (1916), with its restoration by suture at the close of the operation. This lifting of the anterior border of the liver fully exposes the gall-bladder and the cystic and common ducts which are thus brought very near the surface. It may be noted as pointed out by Robson (1904) that, "with the liver in this position, the cystic duct does not form an angle with the common duct, but instead an almost straight passageway is found from the fundus of the gall-bladder to the entrance of the bile-duct into the duodenum."

The ducts are fully exposed to view and with the palpating finger the surgeon may determine readily the condition of the ducts, the duodenum, the pylorus, the pancreas and the regional lymph-nodes.

Very few **special instruments** are required. For the removal of the bile from the gall-bladder a *trocar* and *canula* with a rubber tube attached may be employed. The canula should be large enough to permit the flow of thick, tarry bile and the rubber tube may be attached to a vacuum bottle or a suction syringe may be used. A *spoon scoop*, such as designed by Robson, is of great service in removing calculi from the gall-bladder or ducts. Large and small *gall-stone forceps* should also be provided. The ordinary small teaspoon answers admirably for removing stones from the gall-bladder. A *malleable lead sound*, or flexible bougies, should also be at hand for exploration of the ducts. Retractors, long enough to reach from the surface of the fattest abdomen to the region of the common duct, are indispensable. A malleable retractor about 7 cm. wide and 35 to 45 cm. long is very convenient.

Rubber tubing is needed in all cases of drainage of the biliary tract, either of the gall-bladder or the ducts. We prefer tubing with a lumen of about 0.5 cm. for draining the gall-bladder; the tubing for the ducts should be small enough to enter the lumen of the duct easily, yet large enough to prevent leakage around the tube. A T-shaped piece of tubing is used at times to drain the common and the hepatic ducts as well as for reconstruction of the lumen of the common duct. The senior author has used for some years a specially constructed drainage tube for the gall-bladder. This is made by wrapping a thin layer of gauze around a rubber tube (0.5 cm. lumen), and surrounding this gauze with rubber tissue or preferably rubber dam. The rubber tissue or rubber dam and gauze are held in place by catgut, tied around the whole in several places.

A sterile *hypodermic syringe* always should be part of the armamentarium in gall-stone operations, to be used to identify the common duct before incising it. This precaution of Terrier's (1905) will save the surgeon much uncertainty in cases where the choledochus is dilated and cannot otherwise be distinguished from the portal vein.

Cholecystendysis or *Cholecystotomy without drainage* has been abandoned by most surgeons, as the indications for its use are so seldom seen. The operation was first performed in 1883 by Meredith. Courvoisier performed it in 1884 under the name of cholecystendysis. We think this practice is to be condemned. The supposed indication is to remove gall-stones from the gall-bladder, in the presence of abso-

lutely clear and free cystic and common ducts, and in the absence of infection. It should not be performed if there is doubt about a calculus being overlooked either in the gall-bladder or in the ducts; nor if the lumen of the cystic duct, the common duct or the hepatic duct is even slightly narrowed by inflammatory deposits or cicatricial contractions; nor if the gall-bladder walls or its contents show any evidence, however slight, of infection.

In *exploratory operations* upon the gall-bladder, it may be desired to know the general characteristics of the bile, and without making a formal opening into the organ, a hypodermic needle may be inserted and some of the bile aspirated for study. The opening made by the needle requires no suture.

Operation.—The gall-bladder is exposed and isolated by gauze packs, as in other operations upon the biliary tract. Its fluid contents are removed by aspiration, it is opened, and the calculi removed. The wound in the gall-bladder is then closed with a row of plain catgut sutures which pass through all of the coats of the viscus, and these are reinforced by a continuous Lembert suture, of chromic or iodized catgut. Non-absorbable suture material should not be used for fear of its ulcerating into the lumen of the gall-bladder and forming a nucleus for a stone. The gall-bladder is dropped back into place, the gauze pads are removed, and the wound in the abdominal wall is closed in tiers without drainage. It is always advisable to use two or more interrupted silk-worm-gut sutures running down to the peritoneum through all layers of the abdominal wall ("splint sutures") as advised at page 315.

Cholecystotomy and Cholecystostomy.—The two terms, cholecystotomy and cholecystostomy, are used as synonyms by most authors and writers. On historical grounds it might be preferable to employ the term cholecystotomy to designate solely the operation of opening and draining the gall-bladder as usually done, and to reserve the term cholecystostomy to designate the operation of draining the gall-bladder by attaching it to the parietal peritoneum or anterior sheath of the rectus, as is done when very prolonged drainage is desired (cases of pancreatitis, etc.); but the terms are in such general use as synonyms that the distinction is of questionable value. In many cases the drainage of the gall-bladder is merely incidental to a more important procedure, such as removal of calculi, or evacuation of an empyema; in such cases opening and not drainage of the gall-bladder constitutes the main therapeutic indication. Cholecystendysis is best reserved to describe the suture and replacement of the gall-bladder without

drainage. Mayo Robson, even as late as 1909, still recommended that the gall-bladder should be stitched to the aponeurosis of the rectus whenever possible; only when the gall-bladder is atrophied and shrunken, and cannot be brought up into the abdominal incision was he satisfied to drain it as we advise below.

Operation.—In most cases a simple longitudinal incision 8 to 10 cm. long, through the outer third of the rectus muscle is sufficient. It is wise, particularly in jaundiced cases, to clamp and ligate all superficial vessels as they are cut, to prevent undue bleeding.

The opening in the peritoneum is made large enough to permit proper exposure and manipulation of the viscera. After the wound has been separated with retractors, the field of operation is inspected and explored with the hand. Then a large gauze pad wrung out of hot salt solution is packed toward the mid-line walling off the duodenum and stomach. A second pad of similar character serves to hold the colon and small intestines out of the way; while a third pad is placed in the subhepatic space or right kidney pouch. A fourth pad may be placed between the gastro-hepatic omentum and the left lobe of the liver, and a fifth pad may be passed up over the right lobe of the liver to protect the subphrenic space.

The field of operation is thus walled off from the rest of the peritoneal cavity, and any leakage of bile or infective material will not extend to other parts.

Adhesions should then be looked for, and separated if present. Some will yield readily to the finger, while others must be clamped, cut and ligated. Omental adhesions always should be clamped, cut and ligated, for fear of subsequent hemorrhage.

Now the gall-bladder should be brought into the wound, and if this is not readily done, the right lobe of the liver may be pulled down and rotated out of the wound, as already described, to give a better exposure (Fig. 174). The gall-bladder is then seized at the fundus, and pulled into the wound with the fingers, using a piece of gauze to keep the fundus from slipping away; if this cannot be done with the fingers, forceps may be used. All uncovered surfaces are then protected with gauze.

If the condition is one of hydrops or empyema of the gall-bladder, with marked distention of the organ, it should be emptied by aspiration by means of a trocar and canula. The fluid should be sent to the pathological laboratory for culture. The gall-bladder is then held up by means of forceps and is opened at the fundus with scissors, permitting the insertion of the finger. The interior of the organ is then explored with the finger, the general appearance and condition of the mucosa

and the presence or absence of stones are noted. Stones and inspissated bile are removed by means of scoop, spoon or forceps. Before concluding the operation, unless the patient's condition is critical, the surgeon should examine the common duct and the pancreas. During this examination the gall-bladder is temporarily plugged with gauze. To expose the common duct, the gall-bladder is pulled upon putting the cystic and common ducts on the stretch. The cystic and common ducts may then be examined by sight and touch, the index-finger of either hand being passed into the foramen of Winslow, while the thumb is placed on the free border of the gastro-hepatic ligament. Kehr advised the surgeon to use the left hand, standing with his back toward the patient's head, and keeping his hand fully pronated during this digital exploration of the ducts; but usually we prefer not to change our position, since sight can be used as well as touch. If a stone is present in the common duct, it may be possible to push it along the choledochus into the duodenum; or it may be removed by a scoop or gall-stone forceps passed down into the duct through the gall-bladder and cysticus, if the latter is much dilated. When these methods are ineffective, as they often are, the common duct must be opened in a manner that will be described later (p. 777).

No gall-bladder operation is complete without examination of the common duct and of the pancreas, and searching for enlarged peripancreatic lymph-nodes. At this stage of the operation, the pancreas should be palpated throughout its entire length, the size and consistency of the organ being noted. Enlarged lymph-nodes should be looked for, particularly along the upper border of the pancreas (through the gastro-hepatic omentum), at the junction of the cystic and common ducts and at the junction of the supra- and retroduodenal portions of the common duct.

The gauze is then removed from within the gall-bladder and its interior again explored with the finger. The gall-bladder tube with a lateral opening near its end is then inserted into the gall-bladder for a distance of about 2 cm. and is fastened to the gall-bladder with a catgut suture. A purse string suture of plain catgut, No. 0, is then passed around the gall-bladder about 1 cm. from the opening and left untied (Fig. 175).

The gall-bladder is then grasped with tissue forceps at two opposite points, below the purse-string suture and the tube is invaginated taking with it the edges of the gall-bladder. The purse-string suture is then tied. The advantage of this procedure is that a valve-like closure of the opening in the gall-bladder is formed, so that after the tube is withdrawn the fistula closes rapidly.

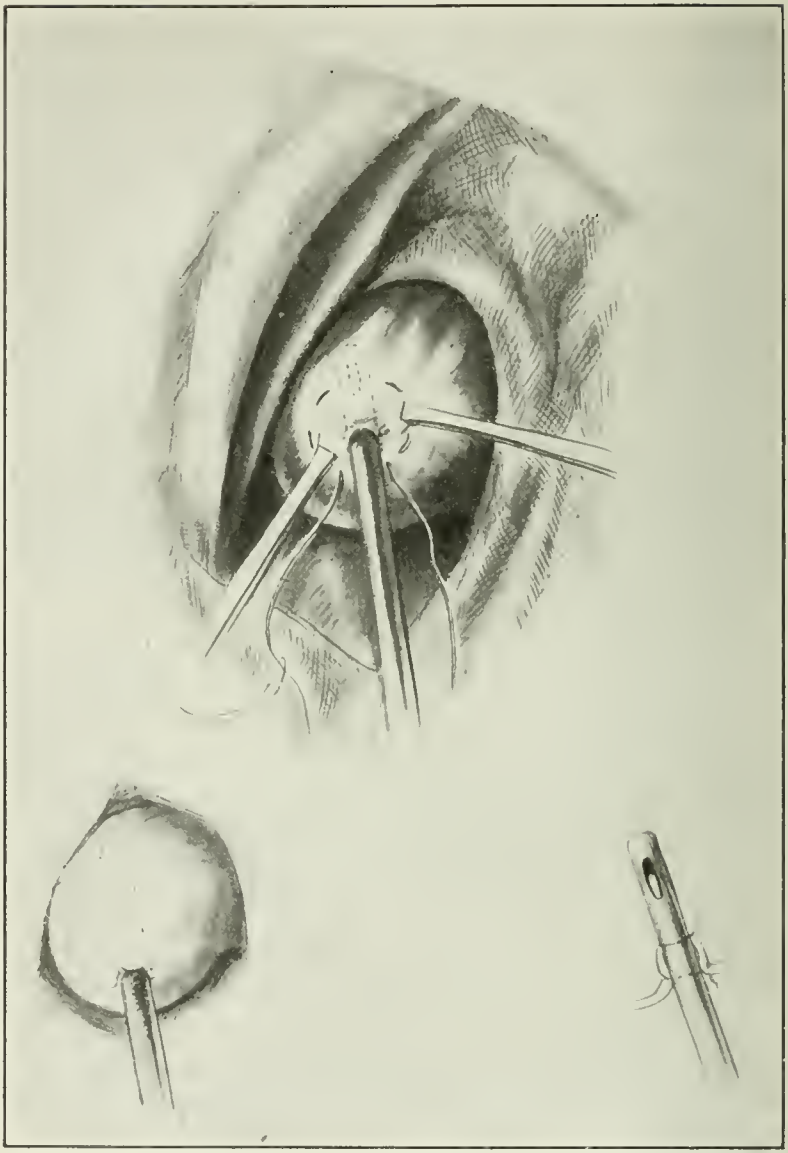


FIG. 175.—Cholecystostomy—Above the Tube is Shown Fixed in the Gall-bladder by One Catgut Suture, with the Purse-string Suture Ready to be Tied. Below at the Left the Purse-string has been Tied. The Diagram at the Right Shows in Section the Inversion of the Gall-bladder Wall.

It may prove impossible to invert a portion of the wall of the gall-bladder in the manner recommended, if the gall-bladder is small or has contracted and thickened walls. Then the opening in the gall-bladder should be sutured as tightly as possible around the tube. In such cases, or whenever there is uncertainty as to the security of the attachment of the tube to the gall-bladder, it is safer to insert also a small cigarette or rubber tissue drain beside the gall-bladder, to provide an exit for leakage. This may be attached by one or two sutures to the fundus of the gall-bladder (Fig. 176). The gall-bladder tube is made of such a length that from 10 to 15 cm. of it project from the abdominal wound or through a stab wound made to the outer side of the former — thus allowing the abdominal wound to be closed throughout. This tube remains in place until it comes away of its own accord, which is usually about the tenth day.

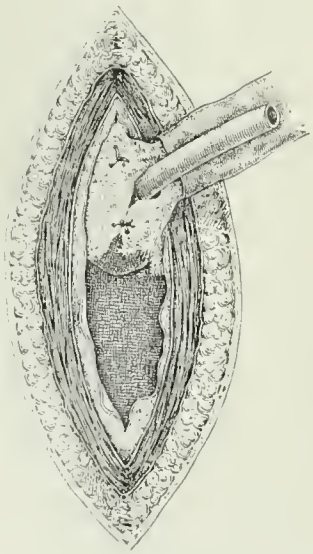


Fig. 176. — Cholecystostomy: Drainage Tube in Gall-bladder and Gauze Ends of Cigarette Drain Sutured to Fundus of Gall-bladder.

All gauze pads and sponges are now removed from the abdomen, the gauze count made and the instruments accounted for. The sand pillow is removed from beneath the patient, as the closure of the wound is facilitated by its removal. The wound is then sutured from its upper and lower angles, toward the point of exit of the tube if the latter is brought out through the wound. The tube is always given exit from the wound where it will afford best drainage; at times, as indicated above, a counterpuncture is necessary to insure good drainage. This depends largely upon the size and position of the gall-bladder. When a separate incision is required for the drainage it usually is made lateral to the operation wound, which may then be closed completely.

The abdominal wound is closed as recommended at p. 314, a dry gauze dressing is applied and is held in place by strips of adhesive plaster and a Scultetus bandage. The dressings are cut so as to allow the drainage tube to protrude, and this projecting tube is then connected by a short piece of glass tubing to a long rubber tube which drains into a receptacle at the bedside; or directly into a bottle fastened within the dressings, which permits a little more freedom of motion in bed.

Cholecytostomy with Prolonged Drainage.—It is especially in cases of cholangeitis, pancreatic lymphangeitis, and chronic interstitial pancreatitis, that this method is advisable. It may require many months for the biliary tract to become sterile.

The *operation* differs in no material respect from that of the ordinary cholecystostomy already described, save in the fact that the gall-bladder is sutured either to the parietal peritoneum or the anterior sheath of the rectus, and is not dropped back into the abdominal cavity after the margins of the opening in its fundus have been inverted around the drainage tube. The other manipulations having been completed, the gall-bladder tube is inserted for about

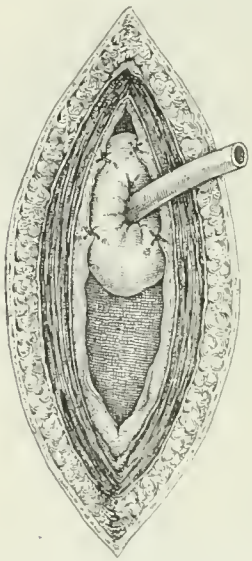


FIG. 177.—Fundus of Gall-bladder Sutured to the Parietal Peritoneum, to Secure Prolonged Drainage.

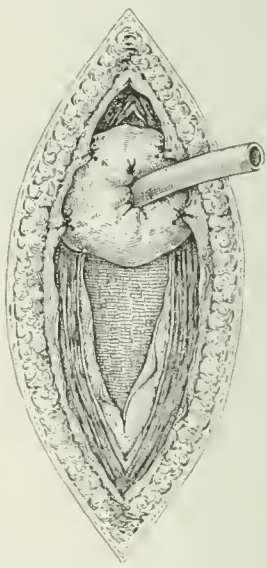


FIG. 178.—Fundus of Gall-bladder Sutured to the Anterior Sheath of the Rectus Muscle, to Secure very Prolonged Drainage.

2.5 cm. into the opening in the fundus of the gall-bladder and is fixed to the margins of this opening with one or two sutures of plain catgut (No. 0). The opening in the gall-bladder is then closed tightly around the tube, with sutures; but if a purse-string suture is used, no effort should be made to invert the edges of the gall-bladder opening, as the purpose of the operation is to secure prolonged drainage and prompt closure of the fistula is undesirable.

When the abdominal wound is being closed, the fundus of the gall-bladder is caught by at least two sutures passing through the parietal peritoneum on both sides of the abdominal incision, so as

to anchor the gall-bladder firmly in the wound (Fig. 177). If very prolonged drainage is desired the gall-bladder is sutured to the anterior sheath of the rectus muscle (Fig. 178). As already noted, Mayo Robson, as late as 1909, still advocated this method of treatment of the gall-bladder, even in cases of simple cholelithiasis, where prolonged drainage is not required; and he recommended only in exceptional cases, where this method proved impossible, the procedure we have described as simple cholecystostomy—dropping the gall-bladder back into the abdomen after closing its opening around a drainage tube.

Cholecystectomy.—In this operation the patient is placed in the usual position, the same incision is made, and the parts are walled off with gauze as described. The best method of removing the gall-bladder is to work from the cystic duct and artery toward the fundus. This is preferable to working in the opposite direction in that the vessels are clamped and ligated first, which is no doubt the most important and at times the most difficult part of the operation. Moreover, when working from the fundus downward to the duct and artery, blood from the denuded surface of the liver may run down and obscure the view, and time is lost in sponging it away.

The liver is pulled down and rotated out of the wound, when this is possible, and is held by an assistant. The fundus of the gall-bladder is drawn taut, and the pouch of the gall-bladder usually over-hanging the cystic duct is caught in forceps and drawn upward, thus making tense the right border of the gastro-hepatic omentum. The cystic duct is next located and isolated after making a small incision through the right free border of the gastro-hepatic omentum, exposing clearly the cystic duct at its junction with the choledochus. By a little gauze or blunt instrumental dissection the peritoneum is wiped away, thoroughly exposing the cystic duct and vessels. The cystic duct, close to its termination in the choledochus, is grasped between two long-handled hemostatic forceps, of which the curved type proves most satisfactory for this purpose (Fig. 179). The duct is then divided between the hemostats. Care must be taken not to include a part of the wall of the common duct; this is best avoided by passing the curved forceps entirely around the cystic duct before clamping it, thus making certain that the wall of the common or hepatic duct is not included in the bite of the forceps. The cystic artery and vein which lie above and to the inner side of the duct are then clamped with two hemostats and are divided between them. At times it will also be necessary to clamp and ligate an anomalous branch of the gastro-duodenal

artery which supplies the cystic and common ducts. The stumps of the duct and vessels may be ligated at this time, or if so desired not until the gall-bladder is removed. In either case iodized or chromic catgut, No. 2, is used. It is a good plan, before ligating the stump of the cystic duct, to explore the common and hepatic ducts as described below. When there is doubt as to the need for drainage through the stump of the cysticus, this may be clamped by a hemostat, as suggested by Ochsner (1906); the hemostat is surrounded by gauze and rubber

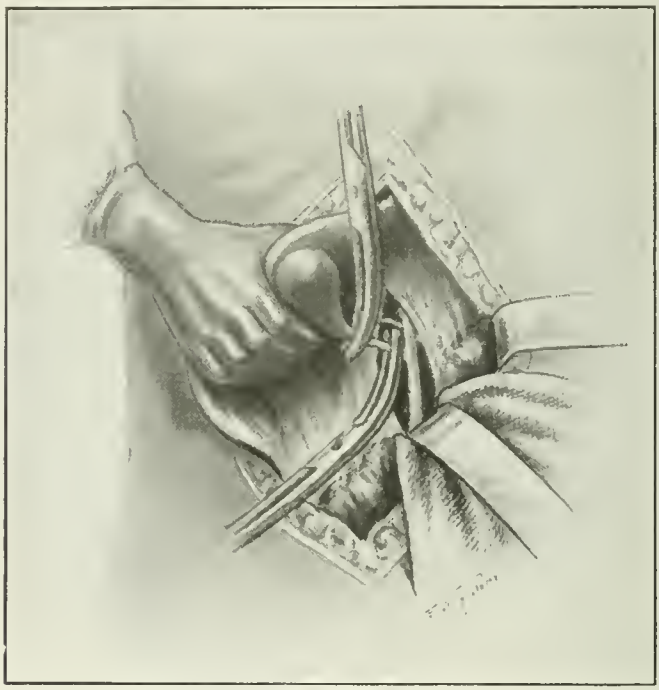


Fig. 179.—Cholecystectomy—the Common, Cystic and Hepatic Ducts and the Cystic Artery are Thoroughly Exposed.

protective, and left protruding from the wound. This clamp may be removed in thirty-six hours, or even sooner if it is decided that drainage is required. Formal drainage is described at page 776. The method of draining the hepaticus after cholecystectomy is described at page 780.

After the stump of the cystic duct and the cystic vessels have been ligated, the separation of the gall-bladder from its fibrous bed is begun. This is done in such a manner as to preserve, if possible, that portion of the fibrous bed immediately adjacent to the liver. By means of the finger the gall-bladder is stripped toward the fundus,

cutting with scissors the peritoneal fold when necessary. When the separation is done carefully, there is very little if any bleeding from the liver substance. The peritoneal fold is then closed by a continuous suture of iodized catgut, unless the hepatic surface is infected, when the gall-bladder bed should be drained. The edges of the opened gastro-hepatic omentum are also closed by suture (Fig. 180). In simple cases the operation is now terminated by closing the abdominal wound around a cigarette drain which extends to the stump of the cystic duct. In no case is drainage omitted, as leakage of bile has sometimes

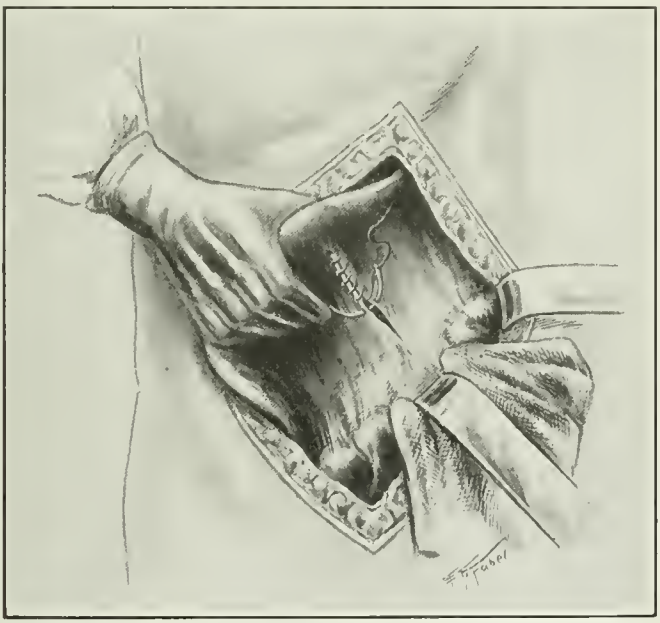


FIG. 180.—Cholecystectomy—after Removal of the Gall-bladder, the Gastro-hepatic Omentum and the Bed of the Gall-bladder are peritonized by Suture.

occurred from slipping or absorption of the ligature. To prevent adhesions between the liver and the pylorus or the duodenum, the omentum should be interposed.

In cases where *very dense adhesions* exist, it may be very difficult to identify the cystic duct before isolating the gall-bladder. Under such circumstances we believe the best technique is that systematized by Terrier: The anterior margin of the liver is identified, and the fundus of the gall-bladder found. The gall-bladder is then opened at its fundus and its inferior wall is cut open little by little by snipping with fine scissors; forceps are clamped on bleeding points in the gall-bladder wall. This incision is continued into and through the cysticus,

right down to the choledochus. This splitting of the cystic duct is the only difficult part of the operation, and it may be very difficult. The duct cannot be distinguished from the outside, on account of adhesions; and it is only recognizable because the operator cautiously follows its lumen, much as one follows the lumen of a strictured urethra in performing external urethrotomy without a guide. When the strictured cystic duct has been split all the way down to the choledochus, the cystic artery is ligated, and the gall-bladder and cystic duct are removed.

The common duct and the hepatic duct are then explored (by different sounds, so as not to carry duodenal infection into the hepaticus), and the hepaticus is drained through the opening left by excision of the cysticus. A rubber drainage tube, or a soft rubber catheter (No. 25 to 33 Fr.), open at the end, but having no lateral fenestrations, is passed up the hepaticus to its bifurcation, where it is felt to be arrested. The tube is then withdrawn slightly, and is stitched in this position, with No. 0 chromic catgut, to the stump of the cysticus, in such a way as not to diminish the lumen either of the duct or the tube. The tube is brought out of the abdominal wound at the most convenient point; and should be carefully distinguished (by color, by the insertion of two safety pins instead of one, or in some other way) from any tube used to drain the subhepatic space.

In cases where the removal of the gall-bladder is attended with much bleeding from the liver substance, this usually may be controlled by suturing tightly together the peritoneal folds which covered the gall-bladder, thus compressing the raw surfaces of the gall-bladder fossa. These sutures, of heavy iodized catgut, should be passed with a large curved blunt needle, and should pass into the liver substance. At times a split rubber tube with a piece of gauze, or a cigarette drain (a rolled piece of gauze surrounded by rubber tissue), may be placed in the fossa, and sutured in place as above described, thus exerting sufficient pressure on the bleeding surface to check all hemorrhage. These drains if used are allowed to remain until they come away easily, with not much pulling, which usually is about the sixth or the seventh day. It is very important in this operation to control all bleeding before closing the wound, and especially is this so in cases with jaundice. Of late we have abandoned the use of the split rubber tube carrying a gauze strip as we find it possible to control the bleeding by sutures carried into and around the gall-bladder bed. In highly infectious cases, however, it is safer to drain the gall-bladder bed. And whenever peritonealization is not complete, the great omentum should

be interposed to prevent adhesions between the gall-bladder bed and the pylorus and duodenum.

After the gauze is removed from the abdomen and the viscera placed in their proper position, it is wise to place a rubber tube in the subhepatic space or right kidney well as far as the posterior abdominal wall. This tube should have a diameter of about 6 to 7 mm. and should be fenestrated near its lower end. The function of this tube is to drain off any blood or bile which may have gravitated to this space during the operation, or which may subsequently collect there. The tube is left projecting from the abdominal wound, or stab wound, for about 3 cm. and through the projecting end a safety pin is passed to prevent the tube from falling into the abdominal cavity. This tube is removed thirty-six hours after operation, and it is not necessary to replace it with any form of drainage.

In cases where there has been considerable hemorrhage or escape of bile into the subhepatic space, it is well to use a glass drainage tube instead of the rubber tube just advised. This tube should be aspirated and turned every twelve to twenty-four hours or oftener if necessary, and should be allowed to remain for thirty-six or forty-eight hours, or until the drainage from it becomes clear and straw colored. When this tube is removed, it is not replaced with any other form of drainage except when the material drained is shown by culture or is believed to be infective; in which case a rubber drainage tube should replace it, and this should be retained until purulent discharge ceases. When a rubber is used to replace the glass tube, it should be passed down through the glass tube and the latter removed by withdrawing it over the rubber tube.

The wound after cholecystectomy is closed around the drainage in the manner which has been described in the previous operations; the tube which drains the hepaticus should not be removed for at least two weeks (page 780); it is best to allow it to remain as long as it will.

Choledochotomy.—Strictly speaking, this term implies merely an incision into the common duct for the purpose of removing a calculus (p. 502); while choledochostomy in its historical sense, indicates suture of the dilated choledochus to the abdominal wound for the purpose of more or less permanent drainage (p. 513). As the former operation (choledochotomy), however, almost always is supplemented by drainage of the duct by tube any distinction between the terms choledochotomy and choledochostomy seems a refinement.

These may be comparatively easy operations or the most difficult

operations in surgery. The difficulties are due to strong adhesions binding the surrounding structures together into a mass which destroys all anatomical landmarks. Especially difficult is the discovery of the choledochus if the gall-bladder and cystic duct have been removed at a previous operation, since there is then no sure guide to follow. The skill of the surgeon will be taxed to the utmost in many cases where the abdominal walls are very thick and the liver is so fixed by adhesions that it cannot be dislocated and brought into the wound. In thin patients, with relaxed abdominal walls, especially women with movable liver, and where no adhesions are present, rotation of the liver is very easy, and the common duct can be brought up into the abdominal incision, permitting all subsequent manipulations to be carried on in full view. In other cases, where the liver is fixed, the difficulties appear to be unsurmountable, but they can be overcome without irremediable damage to the parts, if only proper carefulness and patience are exercised, and the operator possesses sufficient surgical skill. Excellent illumination, and especially the Elliot position, do much to lighten the burden of operating.

Choledochotomy when the Gall-bladder is Present.—The usual incision is made through the abdominal wall, all adhesions are separated, the liver is drawn downward and upward into the wound and rotated. After careful examination of the gall-bladder and ducts has been made for the purpose of locating stones and determining their presence in the common duct, the gall-bladder is tapped and all fluid withdrawn, all precautions against soiling the abdomen, as described at page 768, being carefully carried out. The gall-bladder is then opened and freed from calculi and débris. We believe that this step in the operation is advisable in all cases where the gall-bladder is distended, with a patulous cystic duct, as it will relieve some of the tension in the common duct and prevent excessive escape of bile from the choledochus when this is incised. The position of the calculi in the common duct is then determined by the palpating finger which when passed through the foramen of Winslow may reach almost all portions of the duct. When possible, the calculi should be carefully worked backward into the supraduodenal portion of the duct, if they are found below this portion; or they should be pushed downward into it from the hepaticus, as the common duct can be opened with most facility and safety in its supraduodenal course. It is not an uncommon anomaly for the cystic duct to be inserted at a very oblique angle, and very low in the common duct; thus for some distance the cystic duct runs parallel to the hepatic duct, within the gastro-hepatic omentum.

Trouble may be experienced in some cases in determining the exact location of the common duct and dstinguishing it from the portal vein. We long ago adopted the following method of distinguishing between the two; we are confident it is safe and sure, and we are pleased to know that so skillful an operator as Terrier employed it as a matter of routine in difficult cases. The free border of the gastro-hepatic omentum is carefully incised, a small hypodermic needle is thrust into the lumen of the structure supposed to be the common duct, and the barrel of the syringe is filled with the contents of the structure. The appearance of the fluid withdrawn will show at once whether it is bile from the duct or blood from the vein. The minute puncture immediately closes after withdrawal of the needle; we have never noticed any leakage from such a puncture. When a stone is present in the supraduodenal portion of the duct, it is grasped between the finger and thumb and an incision is made through the walls of the duct, in the direction of its long axis, directly over the stone, and of sufficinnt length to permit easy removal of the calculus. The duct is carefully explored with the finger when the duct will admit its entrance, or with a probe or scoop for the purpose of determining the presence or absence of other stones. When they are present, they should be pushed toward the opening in the duct. When this is not possible they must be removed by one of the methods described below. When the stone is friable, it may be crushed between the finger and thumb, the fragments being extracted with scoop through the original incision in the duct. This procedure may cause too great trauma to the walls of the duct, but we believe that crushing with the finger and thumb, or with the gall-stone scoop, if the proper care is exercised, will do less damage and expose the patient to less risk than the other methods to be described. When the above procedure will not suffice, it may be possible, by the aid of the gall-stone scoop, to push a calculus or several calculi onward into the duodenum.

Thorough exploration of the hepatic duct must be made through the incision in the common duct, the probe being passed into each branch. This probe should be perfectly clean, not one that has been passed before into the choledochus. The probe or small scoop should then be passed into the duodenum for the purpose of determining the patency of that portion of the duct. Occasionally a calculus which cannot be detected in this way may be felt by a finger inserted into the dilated duct. Numerous cases have been reported where calculi have been left behind, repeated operations being required to relieve the patient of stones which should have been removed at the first operation.

Concretions have been left behind by almost every surgeon who has had a large experience in gall-stone surgery, and such cases have been reported by Robson, Riedel, Kehr, Terrier, Fenger, Küster, Lauenstein and numerous others; and both of the present writers have committed similar offenses. Unless all concretions are removed the operation will not afford permanent relief.

After the duct has been cleared it should be drained. If there are no gross lesions in the duct wall, drainage by a tube through the stump of the cystic duct is sufficient, and the opening in the choledochus may be closed with catgut. When the duct is friable or does not seem to be very healthy, or where sutures may not readily be introduced for the purpose of closing the opening in the duct, a rubber tube is passed up into the hepaticus; this tube is held in position with a small chromic catgut suture and the opening in the duct is closed with catgut sutures around the drainage tube, thus preventing leakage of the infectious bile into the abdominal cavity. The tube in the choledochus should be left in place until it comes away of itself. This usually is during the third week after operation. Or, a T-shaped tube may be used, one arm extending toward the duodenal opening of the duct. The latter arm must not be so long as to extend into the duodenum, otherwise some of the duodenal contents will escape by way of the tube, creating a duodenal fistula. The T-tube can be left for three weeks or longer, if thought best. There is no difficulty in removing these tubes after that lapse of time—they come away quite easily.

In almost every case of choledochotomy, the gall-bladder, when present, will have been opened for purposes of exploration and for the removal of calculi, as indicated already. At the conclusion of the operation upon the common duct, the gall-bladder should be drained as described at page 769 (Fig. 175); or if cholecystectomy is indicated the stump of the cystic duct may be employed for drainage of the choledochus.

Drainage of the subhepatic space (page 776) by rubber tube or cigarette drain is indicated in every operation upon the common duct.

Choledochotomy when the Gall-bladder is Absent.—This usually is a difficult operation. A cautious and lengthy dissection may be necessary, to separate adherent stomach, duodenum, omentum, or colon, from the under surface of the liver, and to enable the surgeon, finally having passed these adhesions, to expose the common duct. It is tedious and difficult, but usually possible to expose the supraduodenal portion of the choledochus in this way. When the common

bile-duct has been found, it is opened in the usual way, and the operation concluded as already described. Desjardins (1905) recommended that the operator commence by exposing the choledochus in the retroduodenal portion, by mobilization of the duodenum (p. 784). He thought that in this way time was saved, and there was less uncertainty about the position of important structures. There are cases in which it may seem desirable to incise the duodenum and identify the choledochus by retrograde catheterization through the ampulla of Vater (p. 782). It has been found necessary by the senior author to employ this method only twice among several thousand operations on the biliary tract. The history of one patient was given at p. 518; that of the other is as follows:

OBLITERATION OF CHOLEDOCHUS FOLLOWING CHOLECYSTECTOMY; RETROGRADE CATHETERISM AND DRAINAGE. DEATH

Ella L., aged thirty-six years, admitted to the German Hospital, December 1, 1911. Complaint, jaundice and pain in upper right quadrant of abdomen.

Previous Medical History.—Patient always nervous and never strong. Inflammatory rheumatism at thirteen. Scarlatina at six. Pleurisy at sixteen. Tonsillitis regularly once a year up to fourteen years. Seldom has colds.

Present Illness.—Ten to twelve years ago began to be troubled with pressing sensation in pit of stomach, distention, belching and pain in upper right quadrant of abdomen, coming on about three hours after eating. Sometimes free from pain for four months at a time.

One year ago began to have attacks of severe pain in gall-bladder region, radiating to right shoulder, and sometimes from left costal margin to cardiac region; then sharp pain would radiate over whole body. Had four such attacks. Operation on February 18, 1911—cholecystectomy for gall-stones, also appendectomy. No jaundice or sweats before operation; drained for fourteen weeks following operation. Then well for four weeks. Two weeks after returning home had a second attack of jaundice and nausea. Urine dark, and since drainage has stopped skin has had light yellow tinge. Bowel movements eight or nine times daily. Itching all summer. Two weeks ago had a cold and cough with pain in right hypochondrium and costo-vertebral angle, not sharp and not nauseating. No urinary symptoms. No chronic cough. Some pain in right leg. Condition same during last week.

Physical Examination.—Poorly nourished female. Marked jaundice of sclerae and general jaundice.

Heart: Muscle sound not very strong.

Abdomen: Distended, slight right-sided rigidity. Numbness on pressure over mid-epigastrium. Bimanual palpation reveals large liver, but whether all of tumor mass in upper right abdomen is liver cannot be determined.

Operation.—December 2, 1911. Common duct not found; probe inserted through stump of cystic duct passed only a short distance and became obstructed; unable to find remains of common duct; probe passed upward into hepatic ducts without difficulty. Pylorus isolated; intestines walled off well, the duodenum then

opened; ampulla of Vater found and probe passed, but only a short distance in the lower portion of the gastro-hepatic omentum—probably the distal portion of remains of common duct. A large probe now passed through distal end of common duct into duodenum; still larger probe used in similar manner, but it became obstructed at ampulla. The ampulla was then incised and large probe passed freely. T-tube inserted, one end in distal extremity of common duct and other end in stump of cystic duct; sutured in place with chromic gut. One end of tube passed up through cystic into hepatic duct. Duodenum now closed with iodine gut followed by two layers of continuous linen. One spiral tube drain down to site of operation. Wound closed with chromic gut; two through-and-through silk-worm gut sutures; skin closed with silk-worm gut. Iodine dressing.

Following operation patient complained of weakness although general condition remained good.

Pulse of fair volume. Jaundice much improved.

December 7, 1911.—Slight bleeding from wound which was readily checked by Monsel's solution.

December 8, 1911.—At 9.30 P.M., patient vomited 500 c.c. of dark grumous liquid—looks like blood. Pulse rapid and thready. Abdomen soft, no distention. Fair peristalsis. No evidence of peritonitis or obstruction. Transfused and active stimulation given—patient greatly improved. During the night patient vomited bright blood and expelled blood per rectum. Origin of bleeding looked for but not discovered.

December 9, 1911.—Died 10.20 A.M. Wound opened and stomach found distended with yellow fluid. Site of operation in excellent condition. No evidence of peritonitis. Mesentery of the ileum contained an ecchymosis a little larger than a dollar. Near the mesenteric attachment a distended blood-vessel, apparently thrombotic, stands out prominently. The small intestines contained blood.

Patient evidently died as a result of the hemorrhage which in her greatly weakened condition she was unable to withstand.

Transduodenal Choledochotomy.—This term is applied to the operation of choledochotomy when the calculus is removed from the choledochus through an incision made in the duodenum. The operation was first performed by McBurney, in 1891, followed by Kocher, in 1895, and by Mayo Robson in 1898. It is applicable to cases of impaction of a calculus in the retroduodenal, pancreatic, or interstitial portions of the common duct. The interstitial portion includes the ampulla of Vater, a dilated portion of the duct in which a stone may become lodged and from which it can only with great difficulty be dislodged either into the duodenum or back into the pancreatic portion of the duct.

The abdomen is opened and the biliary passages liberated from adhesions and if possible brought into the wound in the usual manner. The duodenum and the terminal portion of the duct are raised by the fingers of the left hand, and the anterior wall of the duodenum is

incised. McBurney advised a transverse incision, and this was employed by Kehr. We believe that an incision parallel to the long axis of the bowel is preferable, as it gives better exposure of the struc-

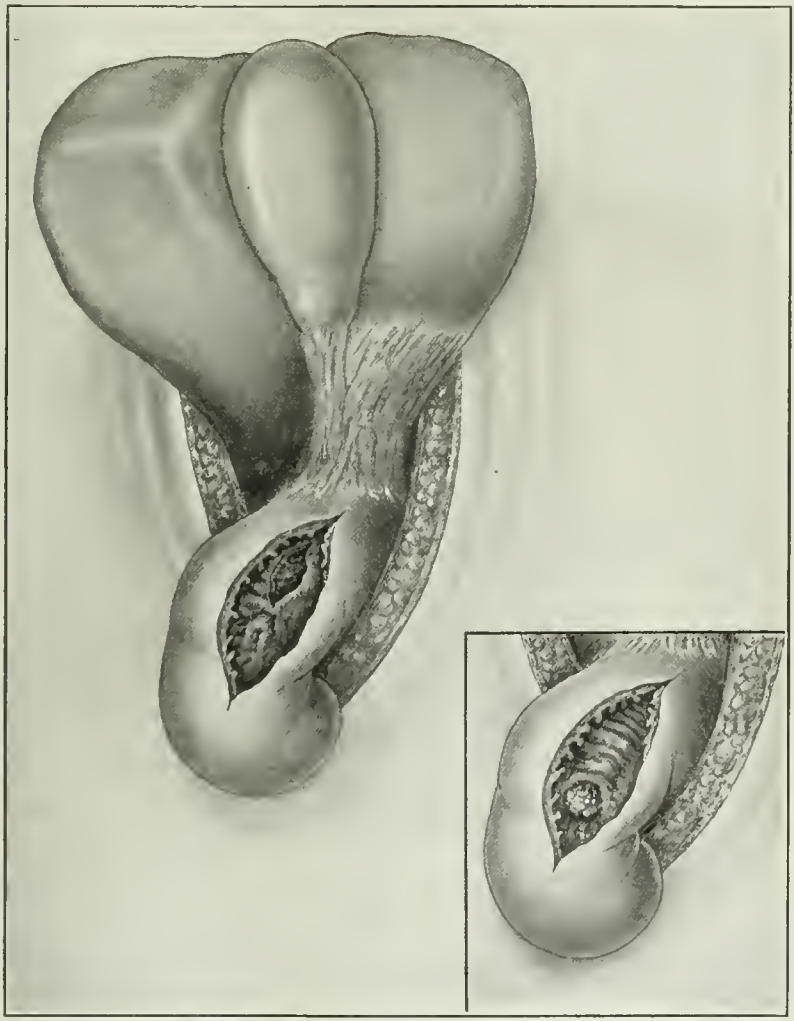


FIG. 181.—Transduodenal Choledochotomy. In the Larger Drawing the Retroduodenal Portion of the Choledochus has been Incised over an Impacted Stone by Means of an Incision in the Posterior Wall of the Duodenum, Exposed by Opening its Anterior Wall (Kocher's Operation). The Smaller Drawing Shows a Calculus Protruding into the Duodenum from the Ampulla of Vater.

tures within the lumen of the intestine and is more easily closed with less danger of leakage or subsequent stricture. After the duodenum

has been opened, the papilla is to be located; if a stone is found within the ampulla the biliary orifice is either dilated or incised upward, and the stone removed. When the stone is lodged in the pancreatic or retroduodenal portions of the duct, the posterior wall of the duodenum may be incised, thus exposing the wall of the choledochus; the stone is then removed through an incision made into the duct directly over the stone. When the papilla has been dilated or incised, no attempt is made to close the opening. When the posterior wall of the duodenum and the common duct have been incised separately it is advisable to suture the opening in the duct to the incision in the posterior wall of the duodenum in such a manner as to form a fistula. This operation was introduced by Kocher (1895) and is named by him *duodeno-choledochostomy*, thus distinguishing it from *duodeno-choledochotomy*, the operation of McBurney.

The incision in the anterior wall of the duodenum is closed with catgut, with a reinforcing suture of linen. If the choledochus has already been opened in its supraduodenal portion for the purpose of removing a stone, it should be drained in this situation; but if it has been opened only by the transduodenal route no external drainage of the duct is necessary. The operation is then completed in the usual manner.

The senior author, in his entire experience, has been forced only twice to adopt the transduodenal method of approaching the common duct which in both cases had been partially destroyed. In all his operations for calculus it has been possible either to extract the stone (entire or after breaking it with the gall-stone scoop) through an incision in the supra-duodenal choledochus or to push the calculus into the duodenum by manipulation with the finger or the gall-stone scoop.

Retroduodenal Choledochotomy.—When a stone is impacted in the retroduodenal or pancreatic portions of the common duct, and cannot be dislodged, this part of the duct may be exposed by **mobilization of the duodenum.** The operation should not be regarded as a substitute for duodeno-choledochotomy which should be reserved for stones impacted in the ampulla of Vater or very close to the duodenal wall. Mobilization of the duodenum is a procedure which consists in separating the duodenal loop from its secondarily acquired attachment to the posterior parietal peritoneum, and restoring it to the state in which it existed during fetal life, with a distinct mesentery of its own (Fig. 5). The right side of the primitive duodenal mesentery becomes adherent to the posterior parietal peritoneum overlying the right kidney; and in extrauterine life this adhesion becomes so intimate

that no visible indication exists of the former state of affairs. If, however, an incision is made just to the right of the descending portion of the duodenum, through the parietal peritoneum overlying the right kidney, it becomes possible by blunt dissection, and with very trifling hemorrhage, to separate the duodenum with its primitive mesentery (which carried the gastro-duodenal vessels) from the original posterior parietal peritoneum, which covers the kidney and vena cava, and thus to restore to the duodenum its former mobility. This is the same principle which is used in mobilizing the sigmoid in resection of the colon, for which operation the method was introduced by Pierre

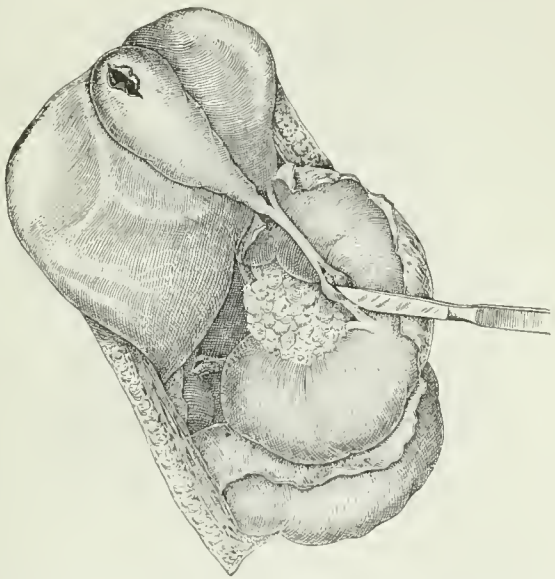


FIG. 182.—Retroduodenal Choledochotomy. The Presence of a Stone Impacted in the Common Duct having been Determined by a Probe Passed from the Gall-bladder or from an Incision in the Supraduodenal Choledochus, the Duodenum is Mobilized, and the Retroduodenal Choledochus Exposed.

Duval (1902). The idea of thus mobilizing the duodenum originated with Terrier, according to Leriche, and was described by his pupil Jourdan in 1895, and by Vautrin in 1896; it is the same method employed by Finney (1902) as an aid in his operation of pyloroplasty; and was systematized by Kocher in 1903, in connection with his operation of lateral gastro-duodenostomy (1892) and his method of partial gastrectomy. (See pages 330 and 351.)

Operation.—The operation is performed thus: after exposing the parts through the usual incision, the descending duodenum is identified. An incision of 7 to 10 cm. long is then made through the fold of perito-

neum just on its right (hepato-duodenal ligament). Into this incision the surgeon inserts his gloved fingers, covered with gauze, and gently wipes the duodenum off the posterior structures, turning it toward the patient's left. As the peritoneal margins are now kept under tension, it is easy to extend the incision upward into the free border of the gastro-hepatic omentum exposing the common bile-duct at the first portion of the duodenum. From this point the choledochus may now be traced downward. It is found to lie either in a groove in the head of the pancreas or to be completely buried in its substance. In the latter

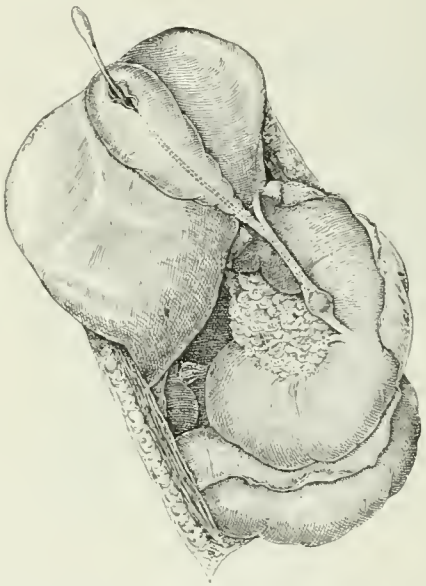


Fig. 183.—Retroduodenal Choledochotomy: the Choledochus is Incised over the Impacted Calculus.

case Vautrin recommends that it be exposed by burning through the overlying pancreatic tissue with the actual cautery. Usually it is sufficient to expose the choledochus by blunt dissection. Bleeding from the lacerated pancreatic tissue may be checked by gauze pressure, or if persistent by suture. The calculus, for the removal of which the operation is done, usually is palpable through the pancreatic tissue and serves as a guide to the exposure of the duct. The duct is to be incised directly over the stone, which is then extracted through the opening in the duct; the entire lumen of the duct is next carefully explored by sound (Fig. 184), or by finger if the duct is large enough. All obstructions having been removed, the opening in the duct is closed

with through and through catgut sutures (or the sutures may include only the muscular and fibrous coats) around the tube which drains the duct (Fig. 185), and the duodenum is replaced in its normal position, the retroduodenal space being drained for two or three days by a rubber tube or rubber dam, to guard against possible leakage from the choledochus. The abdominal wound is then closed in the usual manner.

We have never found it necessary to expose the common duct in this manner.

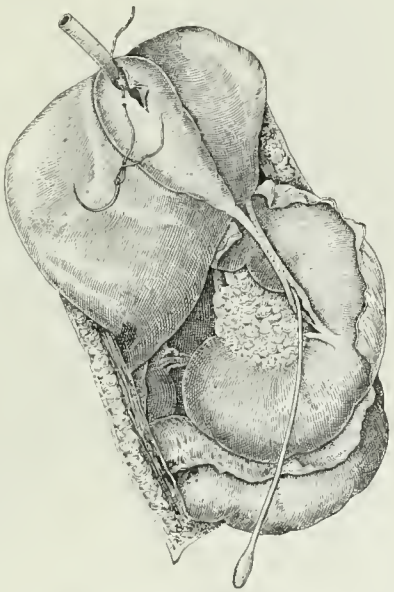


FIG. 184.—Retroduodenal Choledochotomy: after Removal of the Stone, the Hepatic Duct is Explored by Probe.

Cholecystenterostomy.—This is an operation for the establishment of a fistula between the gall-bladder and a portion of the intestinal tract. The duodenum should be selected for the anastomosis when possible; such an operation is termed a *cholecysto-duodenostomy*. If the jejunum is used, it is called *cholecysto-jejunostomy*. Anastomosis with the colon is known as a *cholecysto-colostomy*. We have already (page 515) expressed our preference for an anastomosis with the stomach (*cholecysto-gastrostomy*) whenever the duodenum is not accessible. The indications and contra-indications to cholecystenterostomy have also been discussed at pages 681 and 702.

The anastomosis may be made by direct suture, or by means of a small Murphy button (Fig. 186) or a Robson bobbin. These methods slightly

shorten the time of operation and are more easily performed than the direct suture method, but we prefer the latter because we feel more sure of the firmness of the resulting union of the two viscera. The union when the button is used is very slight immediately following the sloughing of the tissues crushed between the two portions of the button and, as stated by Robson, it is necessary to keep the patient absolutely quiet for two weeks before one can rest assured that the union will not break down and allow escape of the visceral contents.

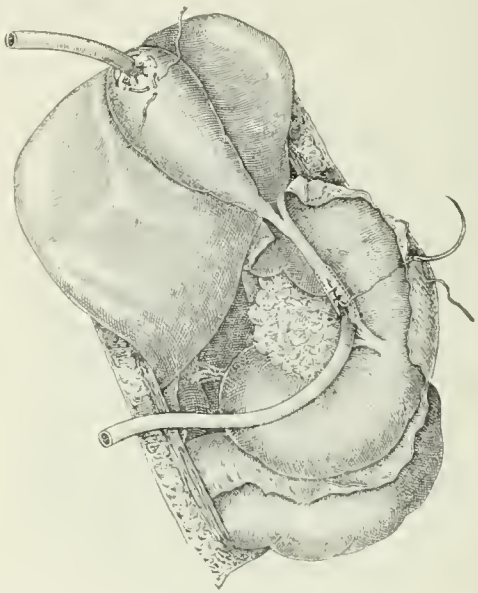


FIG. 185.—Retroduodenal Choledochotomy: Drainage of the Choledochus and of the Gall-bladder by Separate Tubes.

The abdomen is opened in the usual manner for exposure of the biliary passages, and the operative field within the abdomen is cleared of viscera and walled off as previously described. The gall-bladder is opened at its fundus, and is emptied of calculi, bile or débris. The use of a small pair of anastomosis forceps with rubber-covered blades considerably facilitates the insertion of the sutures, but it is not always possible to free the structures sufficiently for their application, even when one adopts the method of mobilizing the duodenum, already described (page 784). Whenever possible, a lateral anastomosis is made by suture, as detailed at page 335. The opening in the duodenum is about 2.5 cm. in length, corresponding in size to that made in the gall-bladder. The anastomosis should be carefully inspected for signs of leakage. The abdominal wound may be closed without drainage but

it is safer to leave a piece of rubber tissue as a drain to the site of the anastomosis.

When the Murphy button is used instead of the suture method, a small sized button is selected. The opening in the gall-bladder is encircled by a running suture of linen. The portion of duodenum or stomach to be used in the anastomosis is freed of its contents by gentle stroking, and clamps with rubber-covered blades then are

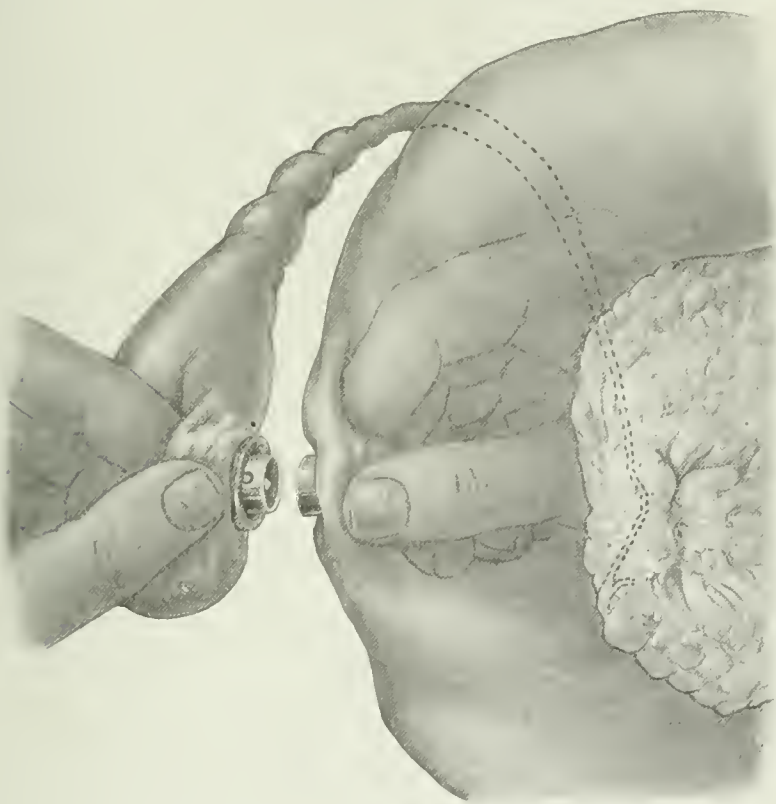


Fig. 186.—Cholecysto-duodenostomy by Means of a Murphy Button.

applied for the purpose of preventing any discharge from the gut during the succeeding steps of the operation. A circular suture of linen thread is then run around the site of the proposed opening in the bowel, and the latter is incised just enough to permit the introduction of one end of the button. The suture is drawn tight around the central barrel of the button, and the second part of the button is fastened into the opening in the gall-bladder in a similar manner. The two ends of

the button are approximated and pushed together firmly. Then it is well to apply a few interrupted sero-serous sutures to hold the viscera in apposition, and relieve the strain on the anastomosis. This completes the operation, and the abdominal wound often may be closed without drainage.

Other methods of biliary intestinal anastomosis, such as **choledocho-enterostomy, hepatico-enterostomy,** etc., have been discussed at page 510.

Operations on the Liver

Hepatopexy.—This is an operation to fix a movable liver. It is very seldom required. Fixation is best secured by attaching the anterior margin of the liver, particularly the right lobe, to the costal margin by means of heavy sutures of chromic catgut. The greater the number of the sutures, the less will be the strain on any one of them.

Operation.—The abdomen is opened by the incision usually employed for operations on the biliary tract, and the proptosed liver is replaced in its normal position. If it is irreducible, it should be fixed in the best obtainable position by means of sutures passed through the muscular and aponeurotic structures of the anterior abdominal wall. When it has been returned to its normal site, the skin and subcutaneous tissues should be dissected back over the costal margin until this is fully exposed. Then a strong round needle, with large curve, and armed with No. 2 or No. 3 chromic catgut, should be passed from above through the costal margin, between the costal cartilages, directly through the costophrenic structures, emerging in the peritoneal cavity close to the anterior margin of the liver. This is then penetrated from above downward, and when the needle has been withdrawn from the under surface of the liver, it is at once carried back again to a point about half a centimetre distant from its original place of entrance, thus completing one mattress suture. From two to eight such sutures should be passed. Injury of the pleura has occurred but has caused no harm. The knots of the sutures thus are placed on the superficial surface of the thorax, just beneath the subcutaneous fat. All the sutures should be placed before any one of them is tied; and just before they are tied the convex surface of the liver and the under surface of the diaphragm should be abraded with gauze so as to promote adhesion. The abdominal incision is closed without drainage. The patient should remain recumbent for three weeks.

Depage's operation (1904) includes, in addition to hepatopexy by a modified technique, also a rather extensive excision of the abdominal wall (*laparectomy*), as he regards laxity of the abdominal wall as one of the main causes of visceral prolapse. The excision involves the whole of the linea alba, and a more extensive area of the overlying skin and fat. By careful suturing of the anterior and posterior sheaths, the two recti muscles (formerly widely separated by the thinned and stretched linea alba) are brought together in the median line, and the umbilical and falciform ligaments of the liver are drawn taut and fixed in the upper angle of the wound.

Other methods of hepatopexy have been referred to at page 539.

Transpleural Hepatotomy for Abscess of the Liver.—A 10 cm. subperiosteal resection of the eighth, ninth or tenth rib (according to the site of the abscess) is done, in the mid-axillary line. It is better to excise a rib that is too low than one that is too high (Fig. 187). If it is thought necessary to remove also a portion of the next rib above, this may be done at the same time, or later. Both ribs may be exposed by an incision over the intervening intercostal space; or, if preferred, a flap of the soft parts may be turned up, exposing two or three ribs. To avoid infection of the pleura the surgeon should next insert, along the lower border of the highest rib exposed, three or four interrupted sutures of chromicized catgut which include the deep layer of the periosteum, both layers (parietal and diaphragmatic) of the *unopened* pleura, and the diaphragm (Fig. 188). This plan, employed by W. W. Ashhurst (1905), and advocated by Körte (1912), is much safer than trusting to inward pressure to hold the pleura in contact with the diaphragm, or than relying on clamps for the same purpose, or even than suturing the two layers of the pleura together after the pleura has been opened, as was originally done by Knowsley Thornton (1886). W. W. Ashhurst, who had a large experience with operations for hepatic abscess during his residence in Mexico, pointed out that it is easy

Fig. 187.—Abscess of the Liver, showing Method of Approach Across Costophrenic Sinus.

to tell by the sense of touch when the needle catches in the diaphragm, and that it is unnecessary to insert these sutures anywhere except as advised, *above* the site proposed for the incision through the layers of the pleura and the diaphragm. According to Körte, Roux recommends

FIG. 188.—Transpleural Operation for Hepatic Abscess; Sutures Passing through Deep Layer of Costal Periosteum, both Layers of Unopened Pleura, and Underlying Diaphragm.

that these sutures be inserted so that each one interlocks with the next. Having thus shut off the pleural cavity, an incision is then made along the upper border of the lowest rib exposed (Fig. 189). This incision divides the deep layer of the periosteum, and both layers of the pleura in the costophrenic sinus. The diaphragm is next incised; this

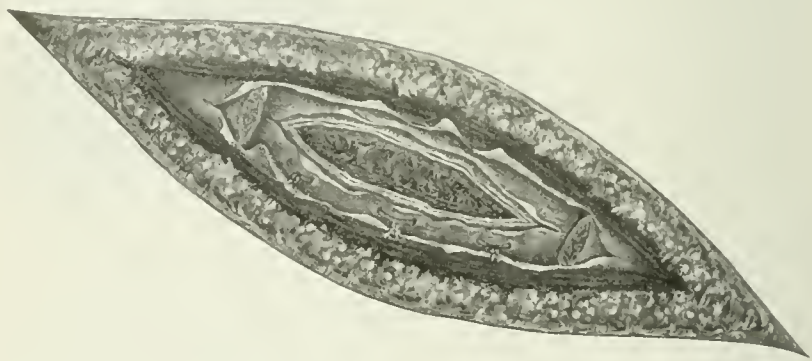


FIG. 189.—Transpleural Operation for Hepatic Abscess: Incision Across Costo-phrenic Sinus.

may be done in the same direction as the former incision, or at right angles to it, splitting the diaphragmatic fibres parallel to their course. The right lobe of the liver may now be freely palpated. The liver, in acute cases, is bluish, soft and pulpy, and often bulges into the wound.

The region of the abscess is more resistant to palpation, and usually is indicated by denser adhesions. Gauze should be packed around the site proposed for the incision into the liver, and if the finger does not break into the abscess cavity, an exploring needle or grooved director should be pushed into the liver. If more room is needed another rib (the next higher) may be excised, but it is not necessary to enlarge the opening in the pleura. When pus is located, an incision into the abscess cavity should be made along the needle or director as guide. For this incision a bistoury is preferable to the actual cautery advised by some surgeons. A free opening for drainage should be secured by dilating the incision with the finger or dressing forceps. Search should be made for neighboring abscesses, which should be opened, if possible, through the walls of the first abscess incised.

Drainage is to be secured by placing two large rubber tubes into the abscess cavity. The remainder of the wound is then tamponed with gauze.

After-treatment.—Irrigation should not be practised for four or five days at the least. It is facilitated by the use of a double tube for drainage, as already advised. These tubes may be shortened from day to day, but not too rapidly. The sinus may take many weeks to close.

Appendicostomy may be necessary if the colitis persists.

Hepatotomy for hydatid cysts has been described at page 567.

Hepatorrhaphy, or suture of the liver, is described below, in connection with hepatectomy.

Hepatectomy.—In removing portions of the liver the main problem is *control of hemorrhage during the operation.* The method proposed by Pringle (mentioned at page 600), which consists in clamping the pedicle of the liver, has not been adopted in the human subject, so far as we know. McDill (1912) thinks the forceps could be kept in place for fifteen or twenty minutes without doing permanent damage.

If the tumor to be excised possesses a small pedicle, which does not itself require excision, this may be *clamped by crushing forceps.* Kocher prefers the use of very heavy crushing forceps which will squeeze all the parenchymatous tissue away from the line of compression, leaving only the capsule of Glisson, which may be easily sutured. Freeman (1919) describes a case in which he removed a cancer of the right lobe of the liver, involving the gall-bladder and transverse colon in one mass; after determining that the growth was apparently primary and that no enlarged lymphnodes could be found, he determined to remove it *en bloc*, together with the cecum, the ascending colon and half of the

transverse colon. (The patient recovered but developed recurrence within six months.) The method of control of bleeding from the liver was simple and efficient:

"In order to get well beyond the tumor it was necessary to resect a large portion of the right lobe of the liver, the line of incision passing through the entire thickness of the organ. Hemorrhage was controlled by tying off the part to be removed by means of two long narrow strips of fascia lata, like pieces of tape. These strips were first pulled directly

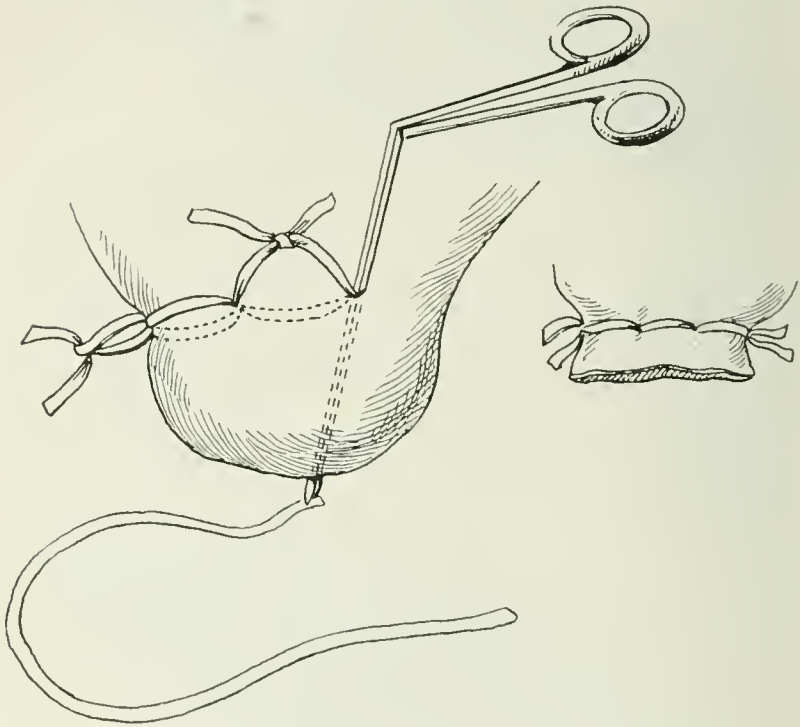


FIG. 190.—Freeman's Method of Resection of the Liver. (*See Text.*)

through the substance of the liver, from behind forwards, with a pair of long alligator forceps, and their respective ends tied very tightly around the hepatic substance to either side, much as one would transfix and tie off the pedicle of an ovarian tumor (Fig. 190). (The presence of the fascia soon checks any bleeding which may occur from the sides of the hole through which it is pulled, provided the opening is not too large; hence the importance of using alligator forceps, or at least those which are long and narrow.) The growth was then cut away, well within healthy liver tissue, without the slightest difficulty or bleeding, in spite of the great thickness of the hepatic stump."

Whatever the method of temporary hemostasis adopted, it is well to make the section of the liver in wedge shape, so as to facilitate closure of the wound (Fig. 191). When the tumor has been removed, the sectioned area requires treatment, for the permanent arrest of hemorrhage and oozing of bile. Ligation of the larger individual vessels should be done whenever possible.

Suture of the liver is difficult only because the usual suture material as ordinarily applied is prone to tear out. The needles employed should be long, rather blunt pointed, and without any cutting edge. For experimental work J. E. Sweet has devised a needle with a female thread at one end, instead of an eye; dry catgut is screwed into this. This plan avoids the "shoulder" formed where the catgut is doubled to pass through the eye of the ordinary needle. Sweet thinks this needle minimizes the traumatism to the liver. Heavy chromicized catgut (No. 2 or 3) is the best material for the sutures, which should be introduced as mattress sutures. They should be tied over a number

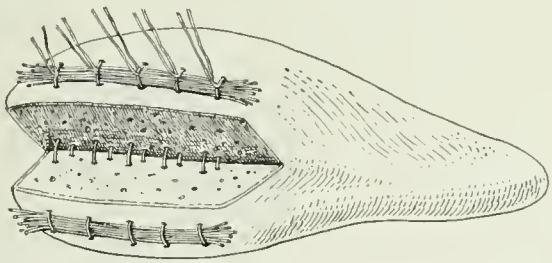


FIG. 191.—Suture of Wedge-shaped Wound of Liver, Left after Resection of a Tumor. The Mattress Sutures are Tied over Strands of Catgut to Prevent them from Cutting out.

of strands of catgut, or a strip of fascia, cut from the abdominal aponeurosis, according to the principle of the old fashioned "quilled suture" (Fig. 191). This plan prevents the sutures from cutting out when they are pulled tight, and makes them efficient in checking hemorrhage. Finally the free margins of the liver flaps should be united with a continuous suture of chromic catgut No. 1.

In all cases drainage of the abdomen is advisable to provide for leakage of blood or bile.

OPERATIONS ON THE PANCREAS

Pancreatotomy for cysts, etc., has been described at page 714.

Pancreatostomy, according to the method of Link, is described at page 687.

Pancreatectomy.—We have already discussed **resection** of the body of the pancreas, and **excision** of its tail (page 701); and it remains

for us here to describe the technique employed for excision of the head of the pancreas, or **total cephalic pancreatectomy,** as it is termed by Sauvé (1908).

The chief indications are carcinoma of the head of the pancreas or of the ampulla of Vater invading the pancreas. As extension to the duodenum from the pancreas, and to the pancreas from the papilla of Vater or elsewhere in the duodenum occurs very early, the complete removal of a tumor in either situation usually involves *pyloro-duodeno-pancreatectomy*. The duodenum, it is true, may be removed without impairing the blood-supply of the pancreas (p. 367), as pointed out by Cotte and Maurizot (1910); but the reverse is not true. Removal of the head of the pancreas requires also removal of the surrounding duodenum, because the blood-supply of the latter is necessarily destroyed.

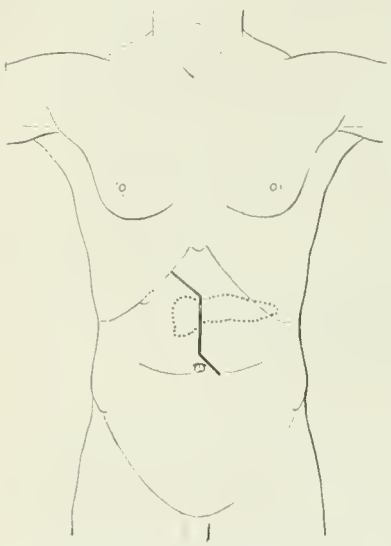


FIG. 192.—Incision for Total Cephalic Pancreatectomy. (*Desjardins.*)

Early diagnosis is necessary to render such an operation of any value; but as noted at page 699 the classical signs of cancer of the pancreas usually are evidences of inoperable growths.

Operation.—Very free exposure is required. The incision recommended by Desjardins (1907) is shown in Fig. 192. A long paramedian incision, or the Czerny incision probably would do as well (page 763).

According to the technique elaborated by Sauvé the operation comprises the following steps: (1) Abdominal incision. (2) Ligation of the pyloric and gastro-duodenal arteries and section of the pylorus. (3) Mobilization of the duodenum. (4) Section of the transverse duodenum, just to the right of the superior mesenteric vessels. (5) Separation of the so-called "little pancreas" from beneath the superior mesenteric vessels, and of the head of the pancreas from the portal vein. (6) Section of the head from the tail of the pancreas; second ligation of the gastro-duodenal artery; ligation and section of the choledochus.

These steps terminate the operation of duodeno-pancreatectomy proper, but there still remain to be done the supplementary operations of gastro-enterostomy and cholecystenterostomy or some of their

modifications. Finally, some disposition must be made of the sectioned tail of the pancreas. Sauvé advises fixing this in the abdominal wound by a method of pancreatostomy somewhat analogous to that employed by Link (page 687). Desjardins suggested that it be implanted into a loop of jejunum; and by means of Coffey's (1909) proposed technique for pancreato-enterostomy, which was successful in dogs, this disposition of the pancreatic stump might be safely accomplished in life.

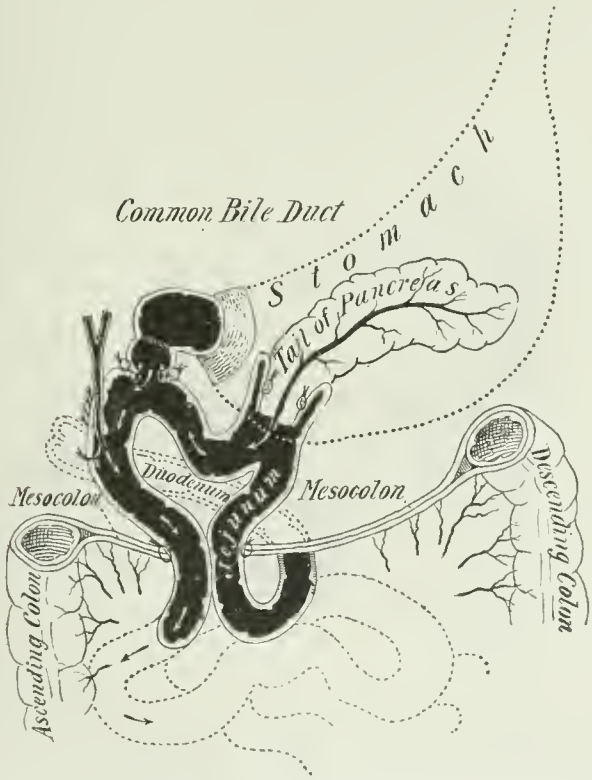


FIG. 193.—Pancreato-enterostomy. After Duodeno-pancreatectomy, the Body of the Pancreas is Implanted into the Apex of a Loop of Jejunum. The Jejunum is Anastomosed with the Pylorus, and the Choledochus is Implanted in it Just Distal to this Anastomosis. (*Coffey.*)

This operation of duodeno-pancreatectomy is so extensive and requires so long a time for its performance that it probably would be better to perform it in two stages. As the most urgent symptom usually is the presence of obstructive jaundice, the first operation might be planned to relieve this by some form of biliary and intestinal anastomosis; but the extremely unfavorable prognosis which attends

such operations for malignant obstruction renders their employment hazardous. It seems probable, therefore, that the radical operations for carcinoma of the head of the pancreas could be indicated only when the condition was found unexpectedly when operating upon a fairly healthy patient. Under such circumstances it would be proper, we believe, to perform at the first operation *posterior gastro-jejunostomy and exclusion of the pylorus* (page 116); at the second operation, two weeks or more later, duodeno-pancreatectomy may be attempted, and in cases of emergency the bile and pancreatic

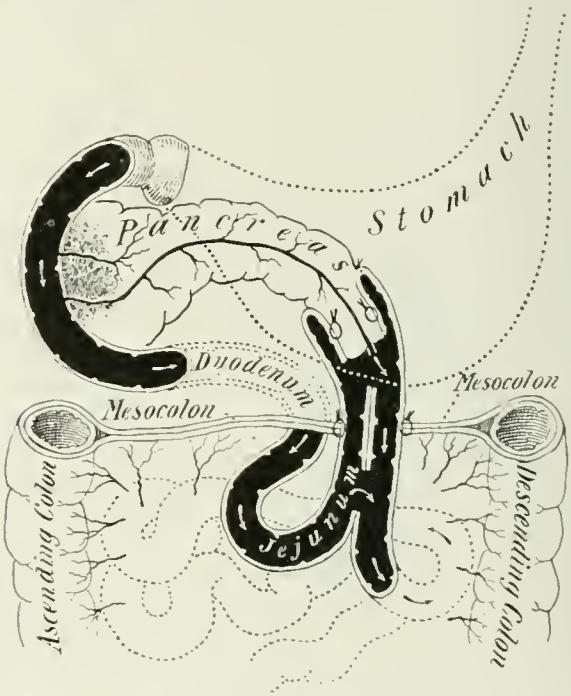


FIG. 194.—Pancreato-enterostomy. As a Palliative Operation the Tail of the Pancreas may be Implanted into the Apex of a Jejunal Loop. A Jejuno-jejunostomy is Also Done. (*Coffey.*)

juice might be drained externally for a time, and the supplementary operations of cholecyst- (choledocho-) enterostomy and pancreato-enterostomy be postponed to a third intervention. These latter operations if done at the first sitting would obscure the operative field so as to render duodeno-pancreatectomy impracticable.

Though such extensive operations on patients so gravely ill seem to offer little prospect even of immediate survival, and still less of ultimate cure, it should not be overlooked, as Sauvé has pointed out,

that even in unpremediated pancreatectomies, done without a well-ordered technique, and with no previous experience, the successes have been more numerous than the failures (9 recoveries among 16 operations). Sauvé concluded that when the surgeon should become possessed of a carefully ordered technique for total excision of the head of the pancreas, and when he should have clearly recognized the indications for the operation, that then the successes would be more numerous, and above all more lasting.

Pancreato-enterostomy.—Desjardins, as already mentioned, proposed to implant the sectioned end of the pancreas into the intestinal canal. The technique of the operation has been worked out by Coffey (1909) in experiments on dogs. He found end-to-end anastomosis was preferable to end-to-side implantation. After section of the jejunum, the continuity of the intestinal tract is restored by implanting the upper jejunal loop into the side of the lower (Y-anastomosis); the open end of the aboral segment of jejunum is then employed for the reception of the sectioned pancreas. Coffey found, however, that the lumen of the bowel was not sufficiently capacious to permit of the intestinal wall being inverted around the stump of the pancreas after the latter had been introduced into the bowel. He therefore adopted the method of throwing the lumina of two intestines into one by making his opening in the bowel at the apex of a loop (Fig. 193). In this way it was possible to obtain a very secure anastomosis. As a palliative operation, Coffey suggested implating the tail of the pancreas into the intestine, thus permitting reverse drainage of the obstructed pancreatic duct (Fig. 194).

Operations on the Spleen

Splenectomy.

Incision.—In traumatic cases the best incision is a longitudinal one above the umbilicus to the left of the median line, as it enables the operator to examine the other viscera. If difficulty is experienced in exposing a spleen which is fixed by adhesions, the left rectus muscle should be divided transversely, as in Czerny's similar incision on the right side for difficult operations on the bile-ducts. In other cases, an incision through the outer border of the left rectus, continued, if necessary, up the rib margin to the ensiform cartilage gives readiest access to the spleen. This is similar to Mayo Robson's incision on the patient's right, for operations on the biliary tract. Sometimes it is easier to reach the pedicle of the spleen from the inner side through a median incision, but usually an incision through the left rectus near its outer border is the most satisfactory.

The abdomen having been opened, the future steps of the operation depend on the presence or absence of adhesions. The latter usually are most numerous between the diaphragm and the upper pole of the spleen although omental adhesions are not uncommon. Before proceeding further, the splenic region should be isolated by gauze pads.

FIG. 195.—Exposure of the Splenic Pedicle through the Gastro-splenic Ligament, after Drawing the Stomach Down and Well Toward the Patient's Right.

The most important part of the whole operation is the *control of hemorrhage*. The capsule of the spleen is easily torn and the pulp is soft and friable. The walls of the veins in the pedicle are thin and tear readily or else are calcareous and brittle. Gentle manipulation therefore is necessary.

In cases where the spleen is not too large, it may be possible to expose its pedicle from the front (Fig. 195), after drawing the stomach far to the right and dividing the gastrosplenic omentum and its contained vessels: the vasa brevia to the stomach and the left gastro-epiploic vessels (Fig. 196). Occasionally the left gastro-epiploic arises from the splenic artery sufficiently proximal to the entrance of the

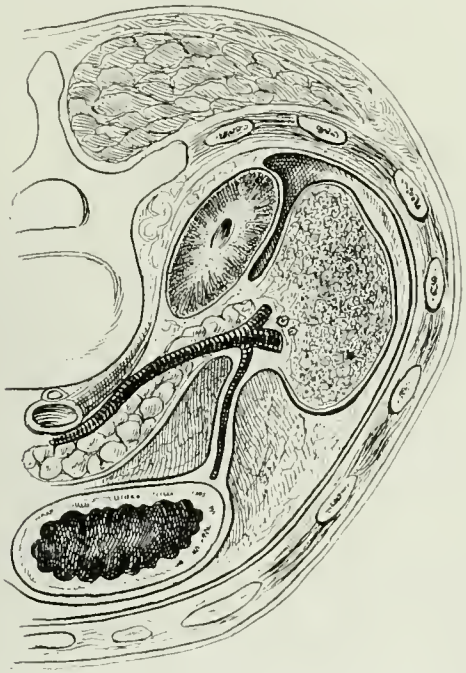


Fig. 190.—Diagram to Show Splenic Pedicle—Transverse Section at Level of Splenic Artery. Note the left Gastro-epiploic Artery (Branch of Splenic) in the Gastro-splenic Ligament.

latter into the spleen as to render it unnecessary to divide the former to expose the splenic pedicle.

In traumatic cases, the splenic artery may be exposed through the gastro-hepatic omentum (Fig. 197), and clamped or ligated in this situation, before the spleen is directly attacked. In most cases, however, the vessels in the pedicle can be more easily reached if the spleen is turned over, but this is not possible until the adhesions are broken down, and the lienophrenic fold of peritoneum divided. These adhesions may not only be very dense, but may contain very large veins which are easily ruptured. Hence it is well whenever possible, to divide the resistant adhesions between two clamps, and as the spleen is gradually mobilized, to pack a large hot moist gauze pad into the bed from which the spleen has been raised. This pack will control

minor venous oozing. When the hand can be introduced finally between the spleen and diaphragm, the former may be drawn down into the wound. It then can be turned over, and the vessels in the pedicle may be isolated by blunt dissection (Fig. 198). If they are not too large they may be secured in two long clamps which should be applied as close as possible to the spleen; the pedicle may then be cut between

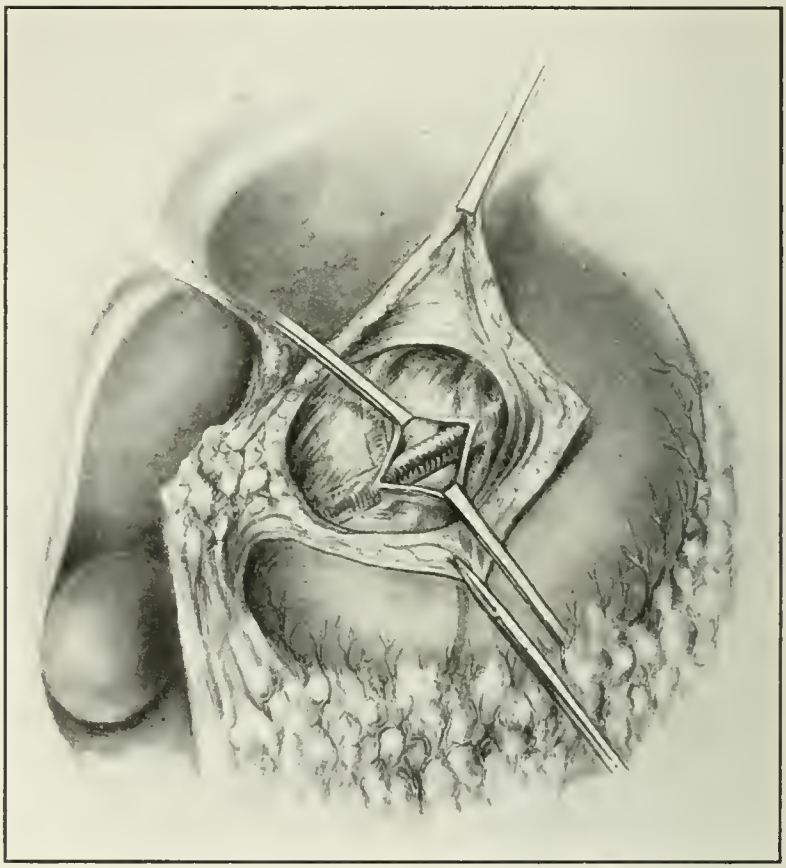


FIG. 197.—Exposure of the Splenic Vessels by an Incision through the Gastro-hepatic Omentum. Note the Celiac Axis Showing through the Posterior Parietal Peritoneum.

them and the spleen and the ligatures applied in the groove made by the deeper clamp before the other clamp is removed. In three out of thirty-one splenectomies, Mayo injured the pancreas, and had to include its stump in his ligatures. All three patients recovered. There must be enough tissue left in the pedicle to admit of the safe application of ligatures and whenever possible each vessel that can be identified should be ligated separately as it projects from the clamps after the spleen has been cut away; these ligatures are in addition

to any ligature used to transfix the pedicle or to ligate it in sections on the proximal side of the clamps. When the bleeding from the pedicle has been stopped the packs are removed and the subphrenic space inspected for bleeding, which must be checked by suture or ligature. Then the abdominal incision is closed in layers in the usual manner. After splenectomy for abscess, or when there is some oozing

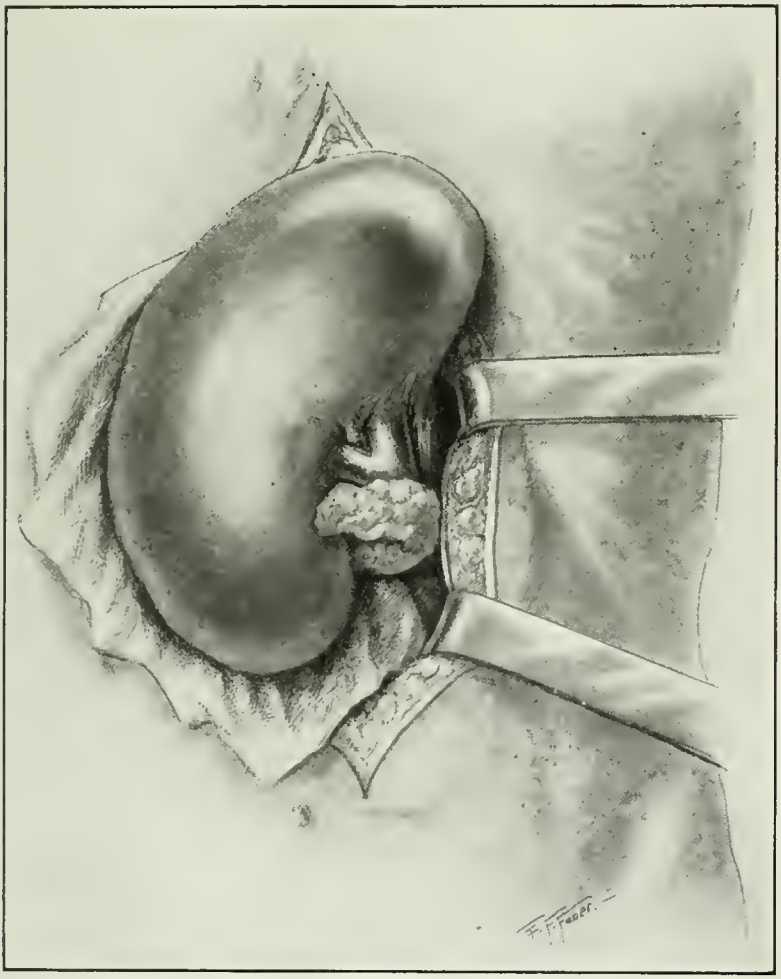


FIG. 198.—Splenectomy—the Pedicle Exposed by Rotating the Spleen towards the Patient's Right. Note the Tail of the Pancreas.

from adhesions, or in traumatic cases, it may be advisable to leave some gauze drainage in the wound. It can be brought out at the lower angle of the incision or preferably through a counter incision in the loin.

Splenotomy.—Cysts and abscesses are the indications for spleno-

tomy and as a rule it is undertaken because splenectomy is difficult or impossible owing to adhesions. Under such circumstances the general peritoneal cavity usually is rather well walled off by the adhesions, but in any case, if exposed, it should be carefully protected with gauze before the spleen is cut into. The abdominal incision may be made over the most prominent portion of the tumor or through the outer third of the left rectus muscle. There are no special points in technique.

Abscesses and cysts that develop backward often can be reached more easily by a transpleural operation, resecting a portion of the ninth or tenth rib in the postaxillary line, sewing the layers of the pleura together to shut off the lung and general pleural cavity (p. 791), and then going through the diaphragm into the abdomen.

The spleen may also be reached through the lumbar incision for kidney operations, if this is extended well forward. On account of mistaken diagnosis this has been done quite frequently.

Splenopexy.—Splenopexy is an operation that is very seldom performed. It is indicated in comparatively few cases, such as those with the spleen normal in size but freely movable, and without any pathological change in its substance or its capsule. These cases occur very rarely.

Moynihan (1908) mentions various methods: 1. Suture of the spleen to the diaphragm or abdominal wall (Tuffier). This is very likely to cause severe hemorrhage. 2. Fixing the spleen in place by surrounding it with gauze to excite the formation of adhesions (Kouwer). 3. Burying the spleen beneath a pocket of peritoneum on the diaphragm (Rydygier). 4. Bringing the spleen out of the abdomen into the lumbar fossa and fastening it there to make it a retroperitoneal organ (Bardenheuer). 5. A case described by Basil Hall in which the lower pole of the spleen was fastened between the layers of the abdominal wound.

Of all these methods practically the only one that is used now is that of packing gauze around the spleen so that it will be held in its normal position by the formation of adhesions.

No fatalities are reported after splenopexy for movable spleen. In most cases the result has been satisfactory, but, as mentioned at page 732, splenectomy was required later in a patient in the German Hospital, owing to increase in size of the spleen.

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